ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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VOLUME XXXI

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THE FURTHER PROCEEDINGS OF THIS INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Good morning, ladies and gentlemen.

MR. LASKIN: Good morning, Mr. Chairman.

DR. DUPRE: Counsel, are there any matters you wish to raise before I raise the witness? Or the parties?

MR. LASKIN: I don't believe so.

DR. DUPRE: May I then, on behalf of us all, welcome most warmly Dr. Allison McDonald...welcome here in two ways - first, her kindness in coming to visit with us this morning, and then more generally welcome Dr. McDonald back to the country because she re-migrated back as of last night.

So welcome indeed back to Canada, Dr. McDonald.

Miss Kahn, would you swear in the witness, please?

DR. ALLISON DUNSTAN McDONALD, AFFIRMED

EXAMINATION-IN-CHIEF BY MR. LASKIN

Q. Dr. McDonald, your curriculum vitae is in at the beginning of the compendium of articles which we have now marked as exhibit forty-two, and I won't go over it but perhaps

Q. (cont'd.) you could tell all of us what your upcoming position is going to be in Montreal, commencing this fall?

A. I am going to head a new team to research in the health of women and their children at work, for the Institute de Recherche sur la Sante et la Securite du Travail au Quebec. It hasn't got an English name as far as I know.

Q. We certainly welcome you back.

A. Thank you.

Q. Could we start your evidence with perhaps one of your most recent papers, and that is the paper you gave at Helsinki on the American asbestos factory studies which is, I understand, in its preliminary stages only insofar as we see it in written form at tab fourteen.

Just so that we all understand what you are looking at, in brief review you are looking at three factories, I take it, one in South Carolina which is the same factory studied by Dr. Dement and which you called factory A in your paper?

A. Correct.

Q. Then factory B, as I understand it, was a factory at Stratford, Connecticut?

A. That's correct.

Q. Has anyone else studied that factory in the literature before, to your knowledge?

A. Not as a separate unit. It was included in one of Dr. Enterline's early studies, but it was not possible to identify the factory separately.

Q. Then thirdly, the factory you call factory C is the factory at Mannheim, Pennsylvania?

A. That's correct.

Q. As I understand it, is that the factory which was previously studied by the NIOSH group of people, including Dr. Lemen, whom we have heard from already, and Mr. Wagner?

A. Yes, that's correct. And earlier by Dr. Mancueso from Pittsburgh.

Q. As I read your article, in terms of the cohort that you looked at the criteria for entry into that cohort was registration by way of a social security number, and employment for more than one month prior to January 1, 1959?

A. Yes.

Q. Is that correct?

A. Correct.

Q. What sort of personnel did that include? Did that include more than people who were actually in the factory or on the factory floor?

A. It included everybody, right up to the president - all persons mentioned in the personnel files.

Q. It could be clerical personnel, secretaries...

A. Yes.

Q. I'm going to come back a little later on to some questions in relation to Dr. Dement's study, but just on this particular point, because as I read Dr. Dement's study he looked at basically employees who worked in the factory for a minimum period of time and I'm wondering whether you could talk to us a little bit about your reasoning for including in your cohort persons who worked anywhere in the operation, be it in the factory or as a secretary or as a president?

A. Well, the philosophy of including a whole cohort, which we had done previously in the chrysotile mines, is that first that if you are making a comparison between that population and an external population there is - which is what is usually done in cohort studies - you have a difficulty over the choice of a reference population. There can be criticisms of any particular population being a satisfactory reference population.

But I suppose the main point in including a complete

A. (cont'd.) cohort is that if you are interested in studying exposure response, it's no use having the more heavily-exposed people alone. You want to have as big a range in exposure as you can in order to give you your relationship between exposure and response. Otherwise you do not...you are not so easily able to detect the relationship.

Q. You have applied that philosophy in Quebec in conjunction with your husband and others who did that study, and also in relation to this study?

A. In relation to this study, yes.

Q. Now, in terms of the fiber type that was used in these three factories, as I read your paper factories A and B used chrysotile, factory C used chrysotile, amosite and some crocidolite?

A. Yes.

Q. Can you tell us how you made...how do you make the determination - or how did you in this case - make the determination as to what factories were using what fiber type?

A. The main...mainly through the records of purchase of asbestos fiber in these factories. It was actually very easy to find out that at the chrysotile plant A, the textile plant that Dement studied, and Dement also had investigated the question of the use of other fibers. With regard to Stratford, the Connecticut brake lining, friction materials plant, again it was, we very easily found out that only chrysotile had been used except fairly recently some anthophyllite had been used in certain products, in a small number of products.

In the factory C, it was always reported that this was mainly a chrysotile factory, but in fact it was generally known that a large amount of amosite was used during the war in order to produce insulation materials, mainly for ships.

A. (cont'd.) It was also known that there was a section at the plant which manufactured crocidolite. In fact, one... it was a very dusty process and when they introduced this process they didn't want to have it with the rest of the plant, so they rented an opera house in downtown...an old opera house...in Mannheim, and they called this the blue. This was generally known as the blue.

But when I got along there to check out what the actual, what the periods were when the different fibers were used, I got them to get out the old lists of purchases and we found that away back in 1925 very substantial amounts both of crocidolite and of amosite were purchased, and obviously used in manufacture because there were subsequent purchases in following years.

So I concluded that from 1925, some of the workers there were exposed to crocidolite and amosite, as well as chrysotile.

Q. I'm looking at the NIOSH article on this particular plant, which you may be familiar with and which we have already put in our record as part of Dr. Lemen's list of articles at tab six, and could I just read to you their description of what they say was the fiber type that was used in this plant, and then perhaps I could ask for your comment on it?

A. Yes.

Q. I'm reading from page 133, and I'll give it to you in a minute.

They say this:

"The plant under study began operations in the early 1900's, and historically used three different kinds of asbestos - amosite, chrysotile and crocidolite. Chrysotile was and is used in all departments in the plant involved with asbestos, which included textile, friction and packing departments. Chrysotile constituted over ninety-nine

Q. (cont'd.) "percent per year of the total quantity of asbestos processed, except for three years during World War II.

During these three years, 1942 through 1944, amosite was selectively used to a limited extent because of naval specifications, and during these three years utilization of amosite increased from one percent to approximately five percent of the total quantity of asbestos used per year, and after the war amosite use dropped to less than one percent. Crocidolite asbestos was processed to a very limited extent in this plant. Its usage was always less than one percent of the total quantity of asbestos consumed, even during the war years".

The reference they give appears to be a personal communication from someone named Weaver.

Do you want...

A. No, I think I know that.

In fact, it was after I had read this paper that I started studying this factory, and I went to Mr. Weaver, who is in charge of environmental control, and it was he who, with digging out the purchasing records, was able to produce this evidence that in fact this had started very much earlier, that it had started in 1925.

I think as far as the point of the amount of amosite and crocidolite, of amphiboles, relative to chrysotile, that one needs to remember that in fact quite small amounts of these amphiboles have caused mesotheliomas.

We have an example in our chrysotile miners study because there was a small factory there, and a very small amount of crocidolite was imported in the war years to manufacture gas masks. It was over two years, and we found a number of mesotheliomas,

A. (cont'd.) I think it was five mesotheliomas, resulting from this one small process using a small amount of crocidolite, so I would say that you should ignore the fact that ninety-nine percent maybe...I don't think it was as high as that... but a large amount of the asbestos processed at Mannheim was in fact chrysotile.

The fact that any reasonable quantity of the other fibers then were processed there creates a completely different situation and it could very well be responsible for mesotheliomas.

Q. When you say the percentages may not be quite as high as ninety-nine percent, but are very high, have you or are you in the process of attempting any breakdown along those lines?

A. Not immediately, although what I am doing at the present time is doing work on the work histories of all the persons who have worked, who were in my cohort, and in the course of that I am going to try to get my colleague who is in the environmental side to work out how many...he will have a record of which processes included amosite and crocidolite, and I hope that we should...we are not going to be able to do a very clear distinction, but we are hoping to make some distinction, and I think at that point we shall have some idea of the relative...certainly of the numbers of employees and possibly of the quantities, but I am not studying that directly.

I have seen, actually, the purchase records, but I'm afraid I haven't calculated the proportion of chrysotile.

Q. I take it that you are presently working on exposure data and quantitative dose-response relationships, although at this preliminary stage in terms of tab fourteen there is no such exposure data?

A. Yes.

Q. Are you working from the same data base as Dr. Dement worked from in relation to his study?

A. Yes, exactly, because that is the data that was available at the plant.

Q. Can you just tell us briefly, because it may help us evaluating Dr. Dement's paper, what is the data source that you do have in terms of exposure information?

A. From three sources. One is the company's own measurements, but for the early period - particularly in the 1930's - a number of surveys by Metropolitan Life, and also some surveys by the government.

Q. If we turn to page four, where you talk about the identification of mesothelioma and your finding that there was only one case in factory A, and I take it from reading your article that it's the same case that Dr. Dement reported; none in factory B and eighteen in factory C?

A. Yes.

Q. Are those death certificate figures, or are those figures derived from going behind the death certificates, as it were, and looking for best evidence?

A. Well, they are not figures derived from the way the death certificates are coded, because on the seventh revision you can't distinguish mesotheliomas from other tumors. They are derived from inspecting, reading through all the death certificates carefully and extracting all those in which a malignant mesothelioma was reported.

I have not done any 'going behind' the diagnosis at the present time, although I hope that I may be able to do so in future.

Q. So that, just so I understand, in terms of the kind of analysis that you have done in Canada and in Quebec in consulting pathologists and so on and so forth, that as yet has not been done in respect of this cohort - or these three cohorts?

A. This is exactly the same as we did in our chrysotile mining study, but when I studied a cohort of workers who had been putting together these gas mask...who had been making these gas mask filters of crocidolite, or putting them together, in that study I went behind the data and I obtained the best information I could about mesotheliomas.

So it's different in those two studies.

Now as far as the ascertainment of mesotheliomas reported by pathologists in Quebec, we have also taken as many of those as we possibly could obtain material and information about and had those submitted to the Canadian Tumor Reference Center panel. So that these all vary slightly in the way in which the diagnosis was confirmed.

Q. In terms of the eighteen mesotheliomas in factory C, do you yet have any information as to whether any or all of those eighteen were exposed at one period of their employment to amphiboles - either amosite or crocidolite?

A. No. No information at all on that exposure, on where they were exposed. Only on the duration of work in the plant.

Q. Just another general question that comes up from one of your other publications where I think you made the observation that in respect of chrysotile exposure and mesothelioma, I take it your judgement is that chrysotile will far less frequently cause mesothelioma. I believe you also observed that the latency period may well be longer, and I'm wondering whether there is any...whether these cohorts later in time, particularly cohorts A and B, may demonstrate some mesotheliomas?

In other words, is there a long enough latency period in respect of most of cohorts A and B?

A. Well, if we are talking about the difference,

A. (cnt'd.) the cases of mesothelioma in the chrysotile miners and millers compared with factories in general, and these factories I am studying in particular, there is not only a big difference in the latency period, but there is also a difference in the site of the mesothelioma. In the chrysotile cohort we only found pleural mesotheliomas. In the factories, we found both pleural and peritoneal mesotheliomas in varying proportions.

But, could I ask you to look at one table which I think might help in this regard, in the paper on mesothelioma in Quebec? Then I'll answer the last part of your question.

Q. I saw a reference to it somewhere myself, I think. Page 676, I think, of tab eleven.

A. Eleven, right.

Q. Page 676.

A. If you could look at table...

Q. Is that the table, table two?

A. Right, 676.

You will see on the bottom line there, the chrysotile mines and mills, these were all sixteen cases of mesothelioma which we have identified as having worked in the chrysotile mines and mills. This excludes the...we had five additional people who had only worked in the factory at Asbestos. They are excluded. They were exposed to crocidolite.

So these are chrysotile only in the mines and mills, and you will see that the latent period there is extremely long.

If you look at the factories in Quebec, where factories B and...factories A, B and C, you will see that factory A was the one at Asbestos, Quebec, and that was a limited exposure to amphibole, to crocidolite. But factories B and C have had an extended exposure to amphiboles, and you will see in

A. (cont'd.) factory B that in fact the number of mesotheliomas was greatest between thirty and thirty-nine years after exposure, and appears to be going down again.

Whereas in the chrysotile mines, the number increases with increasing duration from first exposure.

So I'll say in answer to your question, I think it very unlikely that in the manufacturing setup there may very well be something different in manufacture compared with mining.

I would be very surprised to find cases of mesothelioma later if there hadn't been any earlier.

Q. Is there any biological or medical explanation that you might be able to help us with as to why...well, first of all why the latency period is longer, and second, why chrysotile appears from your studies to produce pleural mesothelioma, and the amphiboles more often peritoneal mesothelioma?

A. I think it's very difficult to be sure about things. I think we have to remember that there is the possibility that these chrysotile cases were not really mesotheliomas. We haven't been able to have some of these studied by the panel.

I think that is one possibility, that a number of these cases are not the same sort of tumor as we see generally. Otherwise, I really...and I'm very suspicious over this question of a difference between pleural...in proportions of pleural and peritoneal mesotheliomas, because there doesn't seem to be any reason why you should not get peritoneal mesotheliomas in heavy chrysotile exposure.

I suppose the answer is that I don't know, but I think there are different, various possibilities to explain this difference, among which is that we are not looking at the same tumor.

That is to say that the mesotheliomas...you see,

A. (cont'd.) not all the mesotheliomas diagnosed are confirmed by pathologists. In fact, in the province of Quebec of mesotheliomas reported by pathologists, of all of them, only fifty percent were approved by the mesothelioma panel, so that you always get a proportion of mesotheliomas that are not really mesotheliomas.

Now, that may differ in different settings, and in an asbestos mining population mesothelioma is likely to have been searched for rather carefully. In fact, we know it has been searched for rather carefully because reports came in 1960 of the South African mesotheliomas, and they started considering whether there were cases in the mines.

DR. MUSTARD: Can I ask a question here?

MR. LASKIN: Sure.

DR. MUSTARD: Do you have an assessment of the history of when the diagnostic capabilities of medicine recognize the importance of mesothelioma and therefore the pathologists, etc., started to apply more rigorous criteria to the diagnosis by jurisdiction, for example, did Quebec become alert and apply a rigorous criteria in the fifties, sixties or seventies and in the United States, in Pennsylvania, did it occur at the same time or later? Do you have any feel for that?

THE WITNESS: No. I think I haven't any feel that there is any general pattern. I think what one can see is that in an area where some cases appear, the pathologists in that area are sensitized and are...become much better in their diagnosis of mesothelioma.

DR. MUSTARD: Could you tell us when you feel that the diagnosis of mesothelioma and distinction from lung cancer became well done in Quebec?

THE WITNESS: I think it is always difficult to distinguish between a peripheral adenocarcinoma, peripheral lung

THE WITNESS: (cont'd.) cancer which spreads into the pleurae, and mesothelioma, and even metastases from some other tumor, unless a very thorough autopsy has been undertaken.

But I don't think that in Quebec generally there is any very good consensus about the diagnosis of mesothelioma. Each pathologist develops his own criteria to some extent, and it is only where you get in groups who see a number of cases that they improve.

I think the whole thing is very hit and miss, and I don't think it has changed.

And I think that it does look as though, the possibility that Quebec pathologists in general have overdiagnosed mesothelioma - based on our experience at ascertaining cases from pathologists, we got a rather high number all over the province of Quebec, but half of those were rejected by this panel of pathologists.

DR. MUSTARD: All right, let me give you an impression I've had, based not on any solid facts. My experience in medicine would suggest in the 1950's, the alertness in ascertainment of mesothelioma would have been at a very low level in general, throughout the people involved in the system which would lead to an emphasis of one form of diagnosis more than another, possibly. But in the sixties, it seems to begin to pick up with a heightened awareness which may have swung the diagnosis the other way, and I'm trying to get some feel as to how that would influence the information we have received about recording the incidence of mesothelioma.

You are in a...

THE WITNESS: I'm sure you are right, but I find it very difficult to see general patterns.

DR. MUSTARD: That leaves me still an uncomfortable feeling, but that's fine.

MR. LASKIN: I'm sorry. Go ahead, Dr. Uffen.

DR. UFFEN: Having interrupted you here, could I ask a little one just for clarification?

I find myself concerned about small numbers, in drawing conclusions from small numbers. I guess you have worried over this.

A few minutes ago I think you mentioned that with respect to factory A, that the rate may be declining in the older age groups because it went from two cases in the age group twenty to twenty-nine, and three cases in the age group thirty to thirty-nine, and then none above that?

THE WITNESS: No...

DR. UFFEN: Have I got that right?

THE WITNESS: No, could I just tell you those are not age groups. Those are intervals from first exposure, and perhaps it becomes clearer if you look at the diagram.

DR. UFFEN: Oh, I'm sorry.

THE WITNESS: The diagram on page 677.

DR. UFFEN: Thanks. I'm sorry. I used the wrong expression.

THE WITNESS: Yes.

DR. UFFEN: Interval from employment.

To take the case between thirty and thirty-nine years, there were three of them in there. What's the possibility that a person was employed thirty-eight and a half years...on the other hand if he had been six months longer, or a little over that, he might have fallen in the class forty to forty-nine years of employment.

You know, a slight shift in the amount of employment would have made it read two, two and one, instead of two, three and zero?

THE WITNESS: Yes.

DR. UFFEN: Then you wouldn't come to that same conclusion, would you?

THE WITNESS: I think you are right in saying that you can't conclude that the cases have declined in the cohort. I would only say that those figures are compatible with mesotheliomas having occurred in the period twenty to forty years after first exposure, and although many people have now been exposed for more than...were exposed more than forty years ago, in fact no more cases appeared.

But you are quite right that you can't really conclude much from those.

I think it's clearer if you look at the diagram on page 677, that these were the periods of employment of those five mesotheliomas, and they all did hit that short period which is marked in the dotted vertical lines, when the crocidolite was used. It fits in, but it is not in itself at all strong evidence.

MR. LASKIN: Q. I'm going to come back, actually, to that study.

Could I just ask you another question for clarification, and it may be I'm behind everybody else in the world, but could you tell us, or tell me in any event, in a little detail what the mesothelioma panel is and who is on it and what its expertise is?

THE WITNESS: A. The Canadian Tumor Reference Center was set up by the Canadian Cancer Society or Association and supported by them. They have set up, as required, a number of panels, particularly for rare tumors, and one of these panels that was set up...I don't know exactly when, but quite a long time ago when the importance of the diagnosis of mesothelioma was made, under Dr. Desmond Magnar, who is now dead, and he invited other pathologists who were interested and had some experience in the diagnosis of mesothelioma to become members of the panel.

A. (cont'd.) So they receive material and information about the clinical history of the case in Ottawa, and they send out similar slides and the copies of the material to this group of pathologists - about six in number - and get their opinion on these cases.

This is without any information about occupational history. It's just on the histological and clinical course.

Now, about three or four years ago one of the world's experts in mesothelioma came as head of the Canadian Tumor Reference Center, and that is Dr. McCawhee. He had a very long experience in England with mesotheliomas, away back to 1960 or so, and he took on this job four years ago and he has done quite a lot of the assessments in this series.

But the expertise of the various pathologists in the group is not rigorously measured, you know, rigorously evaluated. But it is a group of pathologists who acquire experience by getting a lot of cases to review and look at.

Q. Just coming back and finishing the question that we started with, are there any other...apart from the explanation which you have suggested that they may not be evaluating the same tumor all the time...are there any other explanations that you may wish to give us or suggest to us that would explain the pleural/peritoneal dichotomy, or indeed your observation of the longer latency period with respect to chrysotile-induced mesothelioma?

A. No. I think it would be unwise to sort of take the evidence too far. You can only say that when you are looking at it, that we are looking at something which looks rather different because of its long latency and because of the fact that these are pleural tumors and we don't have any peritoneal.

We're pretty sure that we are not missing peritoneal tumors because we have inspected the death certificates very

A. (cont'd.) carefully for the possibility that there were any missed tumors.

As I say, I can only say that they look different and they are not just the same as the other more typical mesotheliomas.

Q. Coming back to tab fourteen for just a minute, do we know what the breakdown of the eighteen mesotheliomas in factory C is, as between pleural and peritoneal?

A. I don't offhand, and I don't suppose I put it in the paper. But a guess, I think it might be about fifty-fifty.

You can tell...the cases are listed in the NIOSH paper by Robinson. Perhaps we could check on that point if you want the answer, because that's probably virtually the same cases.

Q. Why don't we check on it at the break and we'll find it out.

Can I just ask you a few more questions about this study and perhaps in relation to Dr. Dement's paper?

First question I would like to ask you concerns the use of a reference population in relation to calculating standard mortality ratios. Can you, first of all, tell us what reference population you used in calculating your SMR's?

A. I used the reference populations of the States in which the factories were located. Not because I thought those were in each case necessarily the best, but because it was at least a standard way of looking at these three factories.

The fact is that the, Pennsylvania is a very big state and it might well be more correct to relate the factory to one part of Pennsylvania, but it wasn't possible to do this.

Connecticut and South Carolina are rather smaller states and I feel that those are the best populations with which to compare the cohorts.

A. (cont'd.) But no population can be regarded as completely satisfactory, because if you have an employed work force, this is a selected cohort, and you get evidence from healthy worker effect and different patterns of mortality, especially for short-term employees. You can see that the comparison with a general population gives you only one part of a picture. Gives you a picture, but it isn't an absolute thing in any way.

Q. Dement, as I read his paper, used the U.S. rates and he, himself, you may be familiar with it, at one part of his paper lists the various differences between U.S. rates, state rates and indeed contiguous county rates, and it would appear that there are some considerable differences between the United States rate, which is quite low comparatively speaking, and the county rates.

I'm wondering whether you have any judgement or opinion on his use of U.S. rates, and whether indeed that's in part an explanation for the rather striking results that he seems to have obtained?

A. Well, no reference populations, as I have said, are perfect. If he had used the local county rate, he would have got a different relative risk. He would have got a lower relative risk because in fact the county lung cancer rate is high, is very high.

On the other hand, in fact the state...yes, the state lung cancer rate and the U.S. lung cancer rate don't appear to differ very much.

So I wouldn't criticize the use of the U.S. rates, except that I think you could argue that the state rate is more likely to be nearer the truth.

But I do question putting too much weight on this external comparison at all, because you are comparing a working population with a general population and there are all

A. (cont'd.) sorts of differences between those two which might account for differences of an order of one, two or possibly threefold. So that the answer that you get is not an absolute one. You are much better when you can make an internal comparison.

Q. Can I ask you about another couple of propositions that have been put to us during the hearings concerning that paper?

Some question has been raised in these hearings about the conversion ratios that Dement used in doing his quantitative assessment, and I wonder at the early stage of your own study in Charleston whether you have any judgement on that question?

A. No. The conversion ratios that were obtained by NIOSH in I think it was 1968, differed in different departments. The...in the mines we found enormously different conversion factors, doing side-by-side membrane filter and impinger measurements.

I think that the use of a conversion factor of three generally...I think he used six for the preparation department.

Q. Eight.

A. Oh, eight for the preparation department? I think it's perfectly reasonable, I think you could argue that it may be that a higher conversion ratio should be used for some of the departments from the evidence that exists, but perhaps up to six, a ratio of six in some of the departments. But still that doesn't make an immense difference. I don't think it's any good arguing too much about the precise conversion ratio, but rather looking at the difference that would be made by using different ones.

Q. Are you going to be doing that kind of analysis yourself?

A. Well, all I've...the only thing I'm doing at the present time is estimating all exposures in impinger

A. (cont'd.) measurements, and I haven't faced this conversion issue yet.

But I'm analyzing my data in such a way that somebody can say to me, I think the conversion ratio should be this or this for this particular job, and that can be introduced into the analysis.

Q. Can I ask you about one or two other comments that have been about that study? Not yours, but Dement's study.

There has been a methodological concern expressed by one or two of our witnesses in relation to the way Dement calculated his man years at risk, and I may get this wrong, but as I understand it, the concern is that man years at risk are being accumulated in different exposure categories as you go along, as a person goes along and accumulates dust, and then ultimately, of course, will - if he dies - will die in only one exposure category, the highest. So that if you have this particular person contributing man years at risk in all the different exposure categories, the effect would have...may well be to underestimate the risk at lower levels, but overestimate it at higher levels.

A. Well, I must confess that I wasn't aware that that was the case, because the way I...normally people, for each individual accumulate as you say, exposure, but the mortality is calculated by the, time is, the mortality rates for persons alive in a given year and passing through a given period, are obtained from the state. So I don't quite understand how he dies in...what you mean by saying that the mortality is attributed to a particular exposure category that he finishes in.

Perhaps that's a technical point and I haven't understood his anlaysis.

Q. All right. We'll leave the comment there.

The only other comment I think that has been made about the study is the extent to which there may be some

Q. (cont'd.) shipyard exposure with respect to some of these employees. As I understand it, Charleston was a navy port and there was a lot of naval construction and so on, during the war.

Have you found that or has that caused any complications in your own analysis?

A. Well, it certainly has made me think about how to deal with this issue because there certainly was, if you look at the records, there is a good deal of movement between the shipyard and the factory. I think that it has been suggested that one of the reasons for this very high lung cancer death rate in Charleston, the Charleston factory, might be because the people had some prior or subsequent exposure in the shipyard.

I don't think that's a particularly likely thing because I think if you did have that you probably would find you were getting mesotheliomas which you expect to be associated with a shipyard.

It's possible, but I don't see anything to support that, although it's a fact that there is a good deal of...there is some movement. But I think possibly it's only for short periods.

Q. Taken all in all, I suppose when you stand back and look at your own study insofar as it has progressed, and the Dement study, can you give us any help on the extent to which his results of his much smaller cohort are or are not consistent with your own findings to date?

A. My own findings are so far extremely preliminary and they are only based on duration of employment. Such as they are, they do reveal, based on duration of employment, a very high risk of lung cancer and to that extent are compatible with Dement's.

But it won't be until I have worked out the exposure data for my cohort that I will be able to see whether I get a

A. (cont'd.) similar...whether I find a similar exposure-response pattern.

So I would say it was compatible, but in view of the fact that the followup is still incomplete and that I have no exposure data so far, I can't say more than that there is no obvious discrepancy so far.

Q. At this early stage do you have any possible explanations as to what might account for what appears to be a very high relative risk for lung cancer?

A. No. I think that the most likely explanation is that there is something about the textile process which does give rise to a very high risk of lung cancer. It looks as though it is enormously higher than we have found in the chrysotile mines.

Having said it may be the process, we can't exclude some other factor introduced in the manufacturing setting that may account for this difference - some other substance.

Q. A co-carcinogen?

A. Yes.

Q. Is there any suggestion or speculation on what it might be?

A. No. I haven't looked into it much, but nothing comes up immediately.

Q. In terms of the process, are you thinking of or suggesting something to do with the shape or the dimension of the fiber in the mine as opposed to the textile operation?

A. Yes. I would say that it looks as though it's something like that. But also it may be that what one is measuring...you see, we are looking at the response to given environmental measurements, and what is being measured in the mills may be different from what is being measured in a factory because of the further refinement of the fiber.

So it could be that there is just a difference in

A. (cont'd.) measurement, but it also could be that there is a difference in the numbers of fibers which are of a biologically-significant shape as far as lung cancer is concerned.

Q. How do the...in terms of measurements, in terms of the impinger data, how do measurements in this textile operation generally compare as to level with the kinds of dust that you found in the Quebec mines - without becoming very detailed about it? Are the impinger measurements fairly compatible?

A. The instruments are the same and the ways that they were using the instruments the same, or similar. It wasn't a midget impinger, it was a big impinger in Charleston.

Q. What about the levels?

A. Yes. The levels, just to us are extremely perplexing because we have infinitely higher apparent measurements in the mines and in the mills than they have found in the factory, and this is puzzling because in looking at the old pictures of the process in the factory, the thing is covered in a...the room is covered in a cloud and people reported you couldn't see from one end to the other, and yet here we have what is apparently, compared with the milling of chrysotile, a set of very low measurements.

We are just puzzled. I can't go any further on that.

Q. Those are the same...I take it those are the same impinger measurements that Dement had?

A. Yes.

Q. Can we come back to your mesothelioma studies in Canada for a minute, and in particular your study in Quebec in your gas mask workers study?

Can I just ask you one or two questions about the gas mask workers study, which is at tab four?

I take it you looked at three plants, two of which

Q. (cont'd.) were in Quebec, and then a third in Ottawa. Can you tell us a little about the Ottawa plant?

A. The Ottawa plant was where the gas mask...where the filter pads which were manufactured at the two factories in Quebec were put into the gas mask container. It was just an assembly plant.

This was done under wartime conditions. They had to set the thing up very quickly, it was a complete copy of the manufacturing and assembly methods which we used in England at the time.

They had people who worked in one big room assembling these plants (sic) and unpacked the filters and distributed the filter pads among the assembers, and I understand that it was rather a messy, dusty job and there was no control at all.

Q. Was it privately owned or government owned?

A. Government. It was the government...

Q. The federal government?

A. Yes, the federal government, but it was civilian employees, largely. It was a combination of military and civilian employees, but the people who assembled the masks were mainly civilian employees.

Q. Was it only in operation for the specific purpose of assembling these gas masks and after that function was finished did the plant close down or disband its operation?

A. Yes, yes.

Q. Do we...I know you make the point in the article that in relation to this particular Ottawa plant as opposed to the other two plants, the employees were exposed only to crocidolite?

A. Yes.

Q. Are you also, when you say that are you also indicating that in terms of other occupational history there was

Q. (cont'd.) no exposure to chrysotile?

For example, were these employees who worked for two years in Ottawa, could they have been exposed to chrysotile, for example, either prior to or subsequent to their two years work in Ottawa?

A. It's very unlikely, but I also would remind you that if you split off the Ottawa cohort it is really a very small group and we are just dealing with sixteen deaths there.

It is just that the findings in the Ottawa group were compatible, if you look at the pattern, with the findings in the two cohorts who had made these gas mask filters. But I don't think you can argue a very great deal from two cases of mesothelioma in sixteen.

Q. You are now looking at table two on page 343?

A. Yes.

Q. I just want to make sure that I understand this table. The mesotheliomas recorded on the death certificates are under the categories pleural or peritoneal, is that correct?

A. That's correct, yes.

Q. And shows two pleural and two peritoneal?

A. That's correct.

Q. The other five mesotheliomas of the nine then, I take it, are the ones that have the letters A through E beside them, and were as a result of diagnosis by a pathologist subsequent to looking at the death certificates?

A. That is correct.

Q. So in Ottawa there...

A. In Ottawa there were three, I'm sorry.

Q. In Ottawa there were three of them?

A. Yes.

Q. One which was diagnosed as GI cancer, and the third generalized...

A. Carcinomatosis.

Q. Carcinomatosis.

A. That's correct.

Q. Do we have any idea as to how much crocidolite was in fact used in these gas masks? Even in terms of what percentage of the product was crocidolite asbestos?

A. Well, I understand that it was fifty percent of the product, that crocidolite was carded with merino wool and that made a sheet from which the filter pads were cut, and I think it was fifty percent of each.

Q. Do you know, Dr. McDonald, if there is any quantitative exposure information with respect to either the making or assembly of these gas masks?

A. None at all.

Q. There is none at all?

A. No.

In the Nottingham plant they didn't have exposure measurements - the Nottingham, England plant - but they did have quite good dust control. They had some exhaust ventilation, but they had nothing in the plant in Ottawa.

Q. Has there been any risk calculation done in respect of lung cancer deaths in this small cohort?

A. Well, we estimated that very roughly - it can only be very rough - that there were roughly twice as many lung cancer deaths as you would expect in a population of that size.

Q. Okay. Can we then turn to the Quebec study for just a moment, which is at tab eleven?

I take it that it has contained within it the Montreal part of the gas mask worker study in relation to mesothelioma?

A. Yes.

Q. But not the Ottawa...

A. But not the Ottawa plant.

Q. Not the Ottawa plant?

A. Right.

Q. You've got four factories and factory A, is that the factory that...the one in Asbestos...is that the one that is referred to in the Quebec miners study as being the small factory operating in conjunction with the mine?

A. Yes, that's correct.

Q. That's the one?

A. There is some difference in the figures because we subsequently found from, I think one more case, I think there were four cases in the gas mask study that were from that factory, and there are five in that table there.

Q. Can you tell us a little bit about the other factories, B, C, and D, which all appear to be in or around Montreal?

A. Yes. Factory B is the factory...is also included in the gas mask experience, and in fact that is in Montreal, has manufactured only chrysotile, it used chrysotile from Quebec to make mainly friction materials, except for this very short period of less than two years in the war when the government ordered them to or asked them with some pressure to make these gas mask filters.

Q. Is it still operating?

A. The factory is still operating, yes. Immediately after that, in 1942 I think it was, it went back to using just only chrysotile and it has continued only to use chrysotile since.

Q. Has it ever been the subject of a mortality study?

A. No, not as far as I know.

Q. What about factory C?

A. Factory C is in Montreal, and has used mixed

A. (cont'd.) fibers - amosite and crocidolite as well as chrysotile.

Q. What does it make, do you know?

A. Asbestos-cement products, and I'm afraid I don't know beyond that. I can't remember, but I remember the asbestos-cement section.

I think it's mainly...it made insulation and building products for the construction industry, of various sorts.

Q. Is it still operating, to your knowledge?

A. It was still operating eighteen months ago. What has happened to it since, I don't know.

Q. Do you have any knowledge as to whether there has been any mortality study done at that particular plant?

A. There has been a limited mortality study by a graduate student in the Department of Epidemiology at McGill, which has not so far been published so far as I am aware.

Q. Is this the same plant...perhaps you know...is this the same plant that Dr. Becklake and Dr. Gibbs have looked at from the point of view of morbidity and respiratory changes?

A. Yes, I think so. I'm not exactly sure what they did in that plant, but they certainly studied Plant D. They did that when I was in the department, but I think they... Dr. Becklake...could I ask whether Dr. Becklake reported on two plants in or near Montreal?

Q. Yes.

A. One of which manufactured only chrysotile, and one of which manufactured amphiboles as well?

Q. Yes.

A. Well, in that case, that's the factory.

Q. What about factory D? What does it produce? Can you help us with that?

A. I'm afraid I...

Q. If memory serves me, it's a textile plant.

A. I think it's a textile plant. I was going to say that.

Q. Can you just tell us, how did you make the assessment as to what fiber types were used? Was that just a direct communication with the people running the plant?

A. Yes.

MR. LASKIN: Dr. Uffen?

DR. UFFEN: I might make a comment here. I'm not sure whether it's of any use of this case, but when you go back to the Ottawa plant where you said you made extensive inquiries but weren't able to track down...do you recall with whom you discussed this? Is it possible that you sort of left out and didn't know about an area where you might have been able to get the information? This is at the back of my mind.

For example, would you have talked with the people in the National Research Council at that time?

THE WITNESS: Yes.

DR. UFFEN: Would you happened to have talked to Dr. Owen Solant, who became the first chairman of the defense research...and my memory is that they were responsible in some indirect way that I can't recall for that establishment of that plant?

THE WITNESS: Yes. Well, I'm afraid my memory is a bit dim, but we visited various people in Ottawa who had been working for the National Defence Board in running, in creating and running this plant, and working there.

We discovered rather a lot of reluctance on the part of some people to talk about it because it was an official secret, and they thought that even to this day that they might be blamed for discussing what was done.

THE WITNESS: (cont'd.) When you say what information, do you mean exposure information or do you mean information about people who worked there? Because I had difficulty in identifying people who had worked in this plant.

DR. UFFEN: I think you put your finger on the trouble. It's the identification of the people and anything about them, it's probably to this day classified and the people...

THE WITNESS: Yes.

DR. UFFEN: ...which is, in my view, probably ridiculous squared.

If it were sufficiently important enough, I'm not sure whether it is at this stage, you might very well be able to get that information now, Dr. McDonald, by persuading people that it is ridiculous after all these years to have that information classified.

THE WITNESS: Well, I certainly think that it would be useful to get information that was more definitive.

First of all, over the people who were employed it did appear that there were no longer, on the civilian part of that, there were no longer any records that were useful - which could be found.

Now, whether that's correct or not, I don't know, but they insisted...

DR. UFFEN: Or which could be made available?

THE WITNESS: They thought they had been destroyed, but I don't know. But it would be very helpful if we were able to do something more, and certainly I am sure that the cohort that I identified, that a lot more members could be found.

But a few years ago there was a great crisis because it was reported in the Ottawa press about this, and people started getting very nervous, people who had worked there.

THE WITNESS: (cont'd.) At that point people were unwilling to talk about it.

I have got some very good informants among the people who have worked there, but they became nervous about themselves and I'm not sure whether the whole thing...whether it would produce...whether it would cause too much anxiety to produce information. I think it would be very useful if I could get more information.

DR. DUPRE: Dr. McDonald, to your knowledge the asbestos-related deaths would have been compensated, would they not?

THE WITNESS: I don't think they were.

DR. UFFEN: You see, I think some of these people were probably employed under separate employer status, independent of the public service of Canada, and whether or not they would be eligible is a very moot point.

The one observation I would like to make is that I would suspect that the people who do know and who are perhaps unable to unwilling to make the information available, are getting elderly and it will only be another few years before it will be quite impossible to determine it.

So if anything is to be done, it should be done, I think, fairly soon.

THE WITNESS: A Colonel Delsellier, who set up the plant and was the man who got a mesothelioma, through which I found out about this experience, I understand that he got... he worked out the question of compensation for his widow when he knew about his condition, and I understand that he was compensated.

But I don't know of any other people who asked for compensation, or whether they were entitled to it.

There were no other military mesotheliomas that I can remember.

MR. LASKIN: Q. The conclusion of this article that is expressed at page 678, where you say the evidence from this survey continues to support the view that the risk of mesothelioma after exposure to crocidolite is many times greater than after chrysotile, is that still your own judgement on the situation?

THE WITNESS: A. Yes.

Q. Where do you place amosite in all this?

A. Well, I think we are still not sure about amosite. I think we must suspect amosite very strongly, and I think particularly in the light of the mineral fiber content of the lungs of mesothelioma cases compared to the controls that I published recently.

At least I have given...it is in...the article is in press at the present time and has not yet been published.

Q. Is that the one that's at tab fifteen, that you refer to?

A. Yes.

Q. Can I ask you on that point, what kind of weight should one be giving to this kind of analysis, and I ask that question in light of a comment that appears even in that article, and that is that chrysotile fibers may not be retained in the lung very long, and I take depending upon the time when you take the tissue, the lung tissue specimen, depending upon how long a person has left asbestos exposure before he dies, you may not get the most accurate of pictures.

A. Yes, I think that we still...we can't draw conclusions about chrysotile because of the fact that it does look as though it disappears in the course of time.

However, you would, in view of the exactly similar distributions between the cases and what I think were very satisfactory controls, you would have to postulate that they had had exposures at different times, and you would have to postulate

A. (cont'd.) a difference, because there is no reason why there shouldn't be a difference in the exposure patterns of the cases and the controls, and that supposing the cases have been exposed at an earlier period, and a lot of the chrysotile had been leached or dissolved from their lungs, they could have ended up with a similar distribution.

I can only say that this similarity in the distribution is compatible with there not being..with chrysotile not being an important cause of mesothelioma, but it certainly is very far from proof.

On the other hand, you can say that there is clear evidence that amosite is responsible for some mesotheliomas.

MR. LASKIN: I think Dr. Mustard has a question.

DR. MUSTARD: Well, if it's in your field that you are questioning?

MR. LASKIN: Go ahead.

DR. MUSTARD: Thank you.

In this analysis of fibers in the lung, what degree of rigor can you give to the actual distribution of fibers by histological compartment of the lung?

In other words, can you give us an assessment of the measurement of fibers associated with the pleurae versus fibers associated with the finer pulmonary tissue around the alveoli, versus the distribution of fibers in the main bronchi?

Is that being done, and if so with what degree of rigor?

The reason for asking the question is, I would be interested to know what associations you can really get in the pleurae in fibers, whether there might be some differential distribution between the controls and the people who had the mesothelioma.

THE WITNESS: Yes, I think you've got a very good

THE WITNESS: (cont'd.) point. We know very little about mineral fibers in lung tissue and it's a very important area for future research.

You make the point that in fact what we are looking at here, which is really fibers from what is probably a fairly random bit of lung, from cases and controls, and we found a difference between the two.

I think the conclusion I could draw from that would be that the cases have had more exposure to amosite than the controls, because we believe that amosite persists in the lung.

But on the other hand, that doesn't tell you that the amosite got to the pleurae and caused mesothelioma.

DR. MUSTARD: I guess that's the question in my mind. Chrysotile might be clear from the basic lung, but it might not be clear from the pleurae. Yet in doing the analysis it would be difficult to pick that up.

THE WITNESS: Yes. But I think that this tells you something about the exposure of these people, but not about the possible ways in which mesothelioma was caused.

DR. MUSTARD: That leads me to a second question. One of our teachers to the students, who are Commissioners, of course, who have been going through a kindergarten course in these things, one of our teachers says that epidemiological data provides evidence about association...

THE WITNESS: Yes.

DR. MUSTARD: ...and it requires a whole series of steps, I think we were told, to try to provide causality.

But one of the important links to that was the biological data which essentially is derived from basic tissue culture experiments if they are available, and animal experiments.

It seems to me the animal data that we've heard about suggests that all the fibers will induce mesothelioma.

DR. MUSTARD: (cont'd.) Obviously there is a question of how the experiments are done. But one of the questions that occurred to me in listening to this discussion - has anybody done any experiments in animals, that you are aware of, where they have induced mesothelioma with the different fiber types, then allowed the animals to live longer, and then extracted tissues to find out what the residual fiber content of the tissues happens to be?

THE WITNESS: Not that I know of.

You see, I agree with you that you have to have such evidence from experiments as you can, it's important that they are compatible with your epidemiological findings, but I don't think that...I think that epidemiology will take you a certain distance, as you say, in causation, in looking for associations under different circumstances so you get a whole pattern, a whole picture. And I think that it is very important, apart from the association, that you have biological plausibility, not necessarily experimental proof, but biological plausibility.

I don't know quite where the animal experiments get us, because first of all they are largely done by injecting fibers of different sorts into the pleurae where chrysotile causes mesotheliomas the same as glass fibers do, or anything, provided they are of certain dimensions.

I mean, I think that you are asking rather a lot in looking for experimental proof, and we certainly haven't got it.

DR. MUSTARD: I guess maybe my question was simple-minded, as people often are in the kindergarten course in these subjects. It is that in effect if there is a current story, I just was curious if you put chrysotile fibers into the pleural space and got mesothelioma and let the animals live, but the chrysotile fibers disappeared, that would create an interesting biological problem to fit back into the data.

THE WITNESS: I think so, but I think that at the moment the epidemiological studies that are being done, that we can look at the pattern of what we find in the way of mesotheliomas in those and then, as you say, follow it up by further experimental work.

DR. MUSTARD: Let me just ask the question about the loss of chrysotile fibers from tissue. That's essentially based on a combination of observations in human pathological analysis of lungs, plus animal experimentation? Or is it essentially based on animal experimentation?

THE WITNESS: I think it's based on both, but I wouldn't like to swear to it.

DR. MUSTARD: And that would be based on whole tissue analysis rather than selected areas of the tissues? Or do you know the answer?

THE WITNESS: I think it's based on electron microscopy on tissues, not whole lung - pieces of tissue - do support this, do give evidence of leaching of the chrysotile fibers.

MR. LASKIN: Shall we take a ten minute coffee break?

DR. DUPRE: Shall we rise until eleven?

MR. LASKIN: Sure.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: Q. Dr. McDonald, can I just come back to this fiber analysis of the lung question for a moment? Do I take it it's a summary of what you were saying just before the break that that kind of analysis will tell you something about exposure, and something perhaps about retention of fibers, but may not answer

Q. (cont'd.) the question as to whether chrysotile, or indeed any fiber, causes or doesn't cause mesothelioma? Is that putting it fairly?

THE WITNESS: A. I think that there is every evidence that amphiboles stay put in the lung, are retained, and therefore I think that the findings indicate that there was a difference in the exposure between cases and controls as far as the amphiboles were concerned. But I think you can't draw any conclusions about chrysotile because of the problems of clearance.

The figures are compatible with there not being a difference between cases and controls in chrysotile, but you certainly wouldn't be entitled to draw conclusions about difference between chrysotile and amphiboles.

Q. Is there any biological or other evidence as to the importance of retention of the fiber in terms of causing or inducing mesothelioma?

Do we know whether it's important that the fiber stay there once it has interacted with the pleurae, for example?

A. No, we would just be speculating.

Q. Has there been this kind of analysis in relation to lung cancer deaths, or is most or all of the analysis going on in terms of mesothelioma?

A. Yes, I'm not aware of anything going on in lung cancer. One of the problems of lung cancer is that it can be caused...it's very difficult to distinguish the contribution of smoking and of even other factors. Whereas for mesothelioma we know that a substantial proportion of the cases are associated with asbestos exposure.

I think it's easier to study in animals the mesothelioma.

Q. Can I come back more generally to this whole question of fiber type and I take your assessment of it, and I

Q. (cont'd.) wonder if I could ask you a number of questions which relate to arguments that have been made to us over the summer on the other point of view, as it were, from those persons who suggest we ought not to make any distinction amongst fiber types, and I think it would help all of us if we could have your comments on those points.

One point that has been put to us by a number of witnesses - indeed, Dr. Mustard started to raise it just before the break - is the importance or otherwise of the animal experimentation, which as I understand it has included not only injection experimentation, but inhalation experimentations as well.

Can you help us on that? Where do we place that in this whole argument and how much weight should we be giving it?

A. Well, I think that I would always place animal experimentation in a category of information which cannot necessarily be extrapolated to man, and therefore it can tell you something that you can experiment on mechanisms and see whether mesothelioma is produced under different sets of circumstances with different fibers, and so on and so forth. But it doesn't necessarily mean that that is what is going to be happening in man. There may be reasons why fibers don't do the same when they are acquired in a natural way in man, or man reacts differently to them.

So I personally don't feel that...although animal experiments are helpful...I don't feel they are very strong evidence in certain respects.

Q. If we become very specific and talk about chrysotile in respect of animal experiments on the one hand and its experience in the human body on the other, can you suggest what specific differences there may be that support the view that chrysotile operates differently in the human body or in man than it does or apparently does in animal experiments?

A. I think it would be speculating, and you have to remember that glass fiber produces mesothelioma in animals and nobody is suggesting that it is a cause in man.

Q. What is your speculation with respect to chrysotile, or even glass fibers?

A. Well, there are all sorts of things that could be different. Chrysotile behaves differently in the way it splits up into fibrils and the way it fragments. It also, at an earlier stage it behaves differently from the point of view of gaining entry to smaller airways.

I mean, you could suggest...I don't think this is necessarily true...but you could suggest that chrysotile always breaks up into very small pieces of fibril, which maybe by the time they reach the pleurae are not of a length that would produce mesothelioma.

It certainly obviously gets to the pleurae, plenty of it gets to the pleurae, because it causes pleural thickening and is found there in the pleurae.

But it doesn't seem, for some reason, to produce much in the way of mesothelioma.

Q. Just carrying that point a little farther, another parallel argument that has been put to us is yes, that may appear to be the case in the various cohort studies that have been looked at, but one should bear in mind that it may have something to do with the way in which chrysotile has been processed in the past, and if indeed chrysotile is processed differently in the future - as for example produced in finer fibers than in the past - it may well be that we will see the same kinds of effects in relation to mesothelioma as we have seen already with respect to the amphiboles.

A. Well I would have thought that the carding and weaving of chrysotile does fragment chrysotile as much as you

A. (cont'd.) can fragment it, and I don't see that there is any reason to believe in the future that finer fibrils would be created.

You know, I don't think you can take this whole lot of evidence too far. You just have a pattern in which you don't find mesotheliomas in large cohorts of people who worked in chrysotile factories - at least this is just preliminary as far as one of the factories is concerned - but it's more definitive in the Charleston plant.

In the friction materials plant that Berry and Newhouse studied in England, there was no evidence that where exposure was only to chrysotile that any mesotheliomas occurred, and there has been ample opportunity for development of mesotheliomas and it's just that wherever you find the amphiboles have been used, you find substantial numbers of cases of mesothelioma. Just that difference, in my view.

Q. What weight or lack of weight do we give to the fact that in many of these studies there...in relation to mesothelioma...there are no quantitative dose measurements?

In other words, we can't, sitting here in this room certainly can't standardize the various studies in terms of dose. Does that weaken the conclusion?

A. You mean because you get higher exposure levels with amphiboles, often, than you do with chrysotile because it's a harsher fiber and it's more difficult to control the airborne dust?

Q. That's one example, or we've heard a lot of evidence that doses amongst insulation workers are very high, and they happen to use amosite. So that ...but we don't have any measurements, we don't have any quantitative measurements.

A. No, I think it's true, we don't have measurements. But we also know that a lot of workers have been

A. (cont'd.) apparently exposed to a lot of chrysotile because they've got a lot of lung cancer.

I think this is a deficiency in the evidence. I agree.

Q. But not of that magnitude that, at least in your judgement, it would invalidate the conclusion?

A. In my view, there is such a lot of evidence that crocidolite and probably amosite cause mesothelioma that I can't help thinking it would be wise to either limit very severely their use, or eliminate it.

Now, I haven't included chrysotile in it because I think that is a slightly different picture. I would have thought that the conclusion that the Commission must come to is that although nothing can be watertight, that there is a case for more or less banning crocidolite and probably amosite, you perhaps could wait a little for any further evidence to accrue.

Q. Do you make that judgement taking account of chrysotile's relationship to lung cancer? Or do you make that judgement looking only at the question of mesothelioma?

A. I'm just talking about the amphiboles. I'm not talking about chrysotile...about what ought to be done to prevent lung cancer in persons exposed to chrysotile.

I think that such evidence as there is only the manufacture of chrysotile suggests that the textile process has produced a very high risk of lung cancer, and I think that the further continuation of the textile process would have to be very strongly justified.

Q. When Mr. Peto was here, he put forward another proposition on this issue which he asked us to consider, and the transcript will certainly correct me if I've got it wrong, but as I understood his proposition, it was essentially this: That one should not look at absolute numbers in terms of mesothelioma.

Q. (cont'd.) That what one should properly do is look at the excess number of lung cancers, observed minus expected, and look at the ratio of the number of mesotheliomas to the number of excess lung cancers, and if one does that, one sees rather much more consistency amongst the epidemiological studies in terms of different fibers, and I take it his conclusion was that where you are seeing low numbers of absolute mesotheliomas, what you may well simply be seeing is the fact that this particular cohort does not have very much excess mortality - if indeed any - from asbestos exposure.

I think, in fact, you've done some of that kind of analysis in your Cold Springs Harbour paper?

A. Yes. Well, I mean I agree, actually, with Julian Peto that the ratio of mesothelioma to lung cancer in the chrysotile miners is not very different from...it is a bit higher, but at least it is a bit lower as far as mesothelioma is concerned - quite a lot lower - but it isn't out of the same ballpark. You know, it's still within the same ballpark as the other studies.

On the other hand, I think if you look at the more recent evidence, if you...table...

Q. It's your large table, which I think is at...

A. The big table at the back...

Q. There don't seem to be any pages in this...

MR. HARDY: Tab thirteen.

MR. LASKIN: There is tab thirteen, yes. It looks to be the second, third, fourth, fifth page in.

THE WITNESS: Yes. If you look at this ratio where it says O minus...it says respiratory cancer, and then under that, O minus E, divided by mesothelioma, the ratio...where for the chrysotile miners it worked out at four point six.

The two I would just like you to look at are the

A. (cont'd.) Nicholson chrysotile mining cohort, which was a subcohort of our cohort, of the big cohort above, and under chrysotile factory, Dement's paper. In both of those you've got a ratio of seventeen, eighteen lung cancers to one mesothelioma, and we haven't got the...no, we haven't anything else.

But those two are very high ratios of lung cancer to mesotheliomas. On their own, of course, perhaps they don't... they aren't very much, but they are both similar and suggest that it isn't always the same in chrysotile compared with mixed exposures.

DR. MUSTARD: Counsel, may I ask a question?

MR. LASKIN: Yes, please, Dr. Mustard.

DR. MUSTARD: I believe one of the lessons that I think the Commissioners have been learning in this course is that in epidemiology there may be some safety in numbers.

My colleague raised the question about small numbers. If I look in the data in that table from the standpoint of the largest numbers in terms of deaths from lung cancer and cohorts, as I looked at it I got the impression that when you went to situations where the excess mortality from lung cancer was a big number, that you began to pick up some consistency in the ratio.

For example, in the McDonald study at the top you have forty-six excess lung cancers, and your ratio is around four. If you go down to the crocidolite story where the number is fairly high, the Newhouse and Berry one, they have sixty lung cancer deaths, the ratio is four point three.

The next one, Newhouse doesn't show that, which obviously falls out, I suppose from looking at that.

Then when you come down to the insulation, mixed insulation, you get a three eighty-one excess lung cancers in the Selikoff study, and the ratio is two point two.

When you try to look at this having large numbers,

DR. MUSTARD: (cont'd.) because the misclassification problem, as you so clearly showed in the other tab we were discussing, means that if you only get twenty-six lung cancer deaths and one mesothelioma, you only have to flip say five of those lung cancers to mesothelioma to change the thing dramatically.

Have you done an assessment based on numbers, regardless of the kinds of asbestos fibers they were exposed to, and does that show anything? Or can you do that? It may be something that's stupid to do, but taking the safety in numbers question, what happens if you approach all of the information that way?

THE WITNESS: Well, I haven't done it that way. I think that obviously you want to consider what it is you are looking at, what type of exposures, and I think that...I agree with you that those chrysotile cohorts where you get a very large ratio are based on extremely small numbers of...well, they are relatively small excess cancers, they are only of the order of seventeen and eighteen, which isn't very large, and the numbers of mesotheliomas, as you say, might well get very underestimated.

I would take your point, I would agree that I don't...I'm just looking at the general pattern of the way that mesotheliomas are appearing...and the fact, as I said earlier, about the difference in the mesotheliomas in the chrysotile mines compared with the sorts of mesothelioma that you get in the mixed exposures.

It is certainly true that some misclassification could alter the picture.

MR. LASKIN: Q. Another point that has been made to us is that even in the mixed-fiber-type environments, in many of those mixed-fiber-type environments, in fact, the amount of amosite or crocidolite is often quite small and indeed minimal.

Q. (cont'd.) Therefore, we are taking a long leap forward when we place a lot of reliance on the fact that the employees are exposed to more than the chrysotile, in the sense that their predominant and almost exclusive exposure is chrysotile, but there may be some crocidolite or amosite around.

Can you give us any sense as to how we should evaluate those studies where there is that kind of situation?

A. Well, I think we still are rather short of information, but I think that studies of fiber in lung are an important contribution to this question.

We certainly need to know more about the pattern of chrysotile retention to draw conclusions about...and the question of its disappearance in the course of time...to be able to draw conclusions from those experiments....from those studies.

Q. In terms of your own judgement on this issue, and the observations that you've made, what is your opinion as to the reason behind it? Again, we've had various schools on why chrysotile may be acting differently or has different causal effects. One school appears to relate to fiber shape and fiber dimension, and so forth, and we've also heard evidence that there may be different chemical reactions that are going on.

Can you help us, or can you tell us what your own judgement or explanation is for the differences you have observed?

A. No, I don't think I have an explanation.

As I say, all I see is a pattern in which you get a lot of mesotheliomas where you have amphibole exposures, and up to date we have seen very little in the very much heavier chrysotile exposures, and very much greater chrysotile exposures that there have been everywhere.

But I can't offer an explanation on the difference. About the only thing one can say is that the amphiboles have

A. (cont'd.) physical characters which are rather different. They tend to remain as longer fibers once they get into tissues, and to persist like that, whereas chrysotile does tend to split up and to split transversely, too, into much shorter pieces.

But this is all just guesswork as to why there is a difference, which I believe there is.

Q. Fair enough.

In terms of Dement's study which appears in the table and which we have already discussed at some length, I take it from your evidence you feel supports rather strongly the view that you have been taking, can I just go back again to how the analysis was made of the one mesothelioma, and the rest of the deaths.

How were the causes of death, indeed in your preliminary study, and in Dement's, determined?

I suppose what concerns me is your own discussion in several of the other papers about the possibilities for misdiagnosis.

A. Yes. Well, obviously there is the big question mark as to whether cases of mesothelioma have not been detected in Charleston. Now, it so happens, and it's very interesting, that the Department of Pathology at the Medical College of South Carolina in Charleston is terribly interested in mesothelioma and has been trying to detect mesotheliomas for years and years, and it was in that department that Lynch and somebody first described the association between asbestosis and lung cancer in 1935, and they have...from that plant...and they have had a very big interest in trying to find cases of mesothelioma and in discussions with Dr. Russell Harley and other pathologists, they have been looking hard for mesotheliomas, and they haven't found any except this one case

A. (cont'd.) here and one other that didn't appear - that was in a woman and hasn't so far come into either my cohort or Dement's cohort.

So the other side of the question is whether there has been a misdiagnosis into other categories of malignant disease, of any cases that might have occurred. Dement has been through all the records of the pathology department, of malignant disease, that he could extract...or perhaps he has taken out all the deaths in his cohort which had pathology in the department in South Carolina - you can correct me if that's not right - and he has not found any suspicion that there was any...of any possibility that other peritoneal mesotheliomas or pleural mesotheliomas had not been diagnosed.

Q. Was he, or can you tell us, whether he was in fact...was he looking specifically at those persons in his cohort who are no longer alive?

A. He certainly went through all the pathology files in South Carolina - in this department in the Medical College of South Carolina. I can't remember, I'm afraid, whether he just took out his own cohort. But I don't think he did. I think he was looking at the diagnoses and then trying to see whether any of them had been...could have been in his cohort.

But I'm afraid that I wouldn't be sure about, I'm not sure about.

Q. All right.

Do you yourself have any plans for consulting pathologists with respect to the deaths in your own cohort?

A. Yes. I am hoping that I will do something similar - that is to say, search as exhaustively as possible in these areas - in the deaths from malignant disease, certainly all those where there has been an autopsy and where it would be possible to review whether or not the case could have been

A. (cont'd.) mesothelioma.

I hope to do that, but I haven't finished the study yet and I've got to do that first.

Q. I suppose I should ask you whether there will be any further publications out of your own Charleston study in the reasonably near future? I suppose particularly thinking of the lifetime of this Commission, sometime in the next year?

A. Oh, yes. I thought you were going to say the next two months.

No, I certainly hope that I shall have analyses and a report prepared by, say next Easter.

Q. Can I turn to a slightly different topic for a moment? One of the things that you have been kind enough to do is review certain studies by Dr. Finkelstein, which were presented to this Commission, and indeed Dr. Finkelstein has given evidence before this Commission.

I wonder if you could share with us the benefit of your views or opinions or criticisms or comments on either or both of those papers - that is, the morbidity study and the mortality study?

A. The morbidity study was of thirty-five compensated cases of asbestosis, and he used that to try to examine whether there was an exposure-response relationship.

I would not feel this to be a justifiable thing because I think that in making a...in certifying a person for compensation for asbestosis, exposure history is taken into account and therefore it wouldn't be very useful to examine exposure history in relation to that criterion of outcome, of diagnosis.

So that I don't think this contributes useful information on exposure-response.

But when we come to the mortality study, this is

A. (cont'd.) a very shattering set of data in which in persons exposed to, I gather mainly making pipe and using crocidolite, he has found that about fifty percent of fifty-eight deaths were due to either mesothelioma or lung cancer, and this, of course, is very high and is only really paralleled by our own gas mask study. Where he had nineteen percent, he had eleven cases of mesothelioma out of fifty-eight deaths in men who had worked in this plant. We had nine cases of mesothelioma in fifty-six deaths in the total cohort of persons involved in the gas mask procedure, and this was men and women.

In the lung cancer, as far as we could estimate, we had about...very, very roughly...about double the number of cases of lung cancers that would have been expected in the general population.

In his fifty-eight cases...I can't remember the exact number...but it was about thirty percent were...in fifty-eight deaths, thirty percent were lung cancer.

But in particular the mesothelioma picture is very dramatic, and I think that given that you have all these cases of mesothelioma - which I would attribute to the crocidolite exposure - you can't begin to calculate any exposure, useful exposure response because you've clearly got a mixture of crocidolite and of chrysotile, and clearly this is causing a very large amount of, a large number of deaths from mesothelioma and lung cancer.

Therefore again, I don't think it's useful to calculate...it doesn't mean anything to calculate an exposure-response relationship.

I would also criticize the exposure-response relationship calculated in that only persons employed nine or more years were included in the analysis, and as I tried to say earlier that if you are looking for exposure response, you want to have as big a range of exposures as you can, and he should include

A. (cont'd.) people who work for shorter periods of time.

But even so, because of the large number of mesotheliomas I don't think...I think you can say that this is compatible with other situations in which there has been exposure to crocodilite and that that looks like the most plausible explanation for the findings.

Q. What sort of comparable situations are you thinking of, apart from the gas mask workers?

A. Well, I think that it's the gas mask one particularly, but I think that the results don't appear to differ too much from the mixed exposures in the Newhouse and Berry factory, given that the numbers are relatively small.

And possibly...no, I would only go, without looking at it a little more carefully, I would only go so far as that - to say like the gas masks here in Canada and in England.

DR. DUPRE: Could I just follow up on the gas mask/Finkelstein study parallels?

Dr. Finkelstein, of course, excludes from his mortality cohort all individuals who were employed, as I understand it, for less than nine years.

Now, your gas mask study, if I remember - by definintion - studies a cohort whose employment duration was three years....or thereabouts. Three years or less?

THE WITNESS: Yes.

DR. DUPRE: Is that correct?

THE WITNESS: Yes.

DR. DUPRE: So would it not be so that if I was trying to make a direct comparison between the gas mask study and the Finkelstein study that the first difference that I should bear in mind is the nature of the cohort?

THE WITNESS: Yes. Well, I think that one of the questions that doesn't get satisfactorily answered is whether there is an exposure-response relationship for mesothelioma.

But if you accept that there probably is an exposure-response relationship, I would agree that because he has only studied people who worked nine years or more that that might account for the very high mesothelioma, proportion of mesotheliomas.

Yes, I would agree with that.

DR. DUPRE: Now, in terms of the evidence that Dr. Finkelstein was able to give us on total mortality from mesotheliomas in that plant, the latest count apparently is sixteen, that gets you right down to the shortest, to very short periods of unemployment.

At this point, again, to make a comparison to other studies, of course, we would have to know the total number of employees with whatever it is - two months, or one month, or whatever, or more - of exposure.

At that stage of the game you might be able to make some comparisons, say to your gas mask situation more reliably? Insofar as your gas mask study by definition has as its cohort the total number of individuals who were employed, albeit by definition for a shorter duration?

THE WITNESS: Yes. And you could also, of course, make a comparison with the chrysotile factory in Charleston, Dement's factory. I'm just saying that the thing that hits you is it looks rather similar to the gas mask picture, but I agree that they have had different exposures.

DR. DUPRE: On pursuing that very same question, Dr. McDonald, I think it was in your...or was it...in your study at tab fourteen? No.

MR. LASKIN: Three.

DR. DUPRE: I think it's tab eleven that I'm looking

DR. DUPRE: (cont'd.) for here. Do you have any comment about the extent to which Dr. Finkelstein's study bears comparison or otherwise to, at page 676, your factory C in Montreal?

The one thing that I note there is that of course both plants are founded the same year, so you are getting an initial large number of employees who move in.

Factory C, as I understand it, of course, is the mixture of chrysotile and crocidolite...

THE WITNESS: And amosite.

DR. DUPRE: And amosite?

THE WITNESS: Yes.

DR. DUPRE: And it's an A-C pipe plant.

THE WITNESS: It's not pipe, I don't think. I'm very sorry, I don't know what they manufactured. It's...I don't know whether...can anybody be asked to supply information? It's Atlas Asbestos in Montreal.

DR. DUPRE: Now, when I look, you see, at your factory C, and bear in mind not only Dr. Finkelstein's overall study but the evidence that he has given us about individuals outside his cohort..of course if I was trying to make the direct comparison then I would have lots of pieces of information missing, but I'm looking at your table two.

I have the same year started - 1948; number of employees to 1977, of course that I wouldn't have because his cohort is different. Maybe I could get it.

Now, it is the case, however, that...well, this is not directly recorded in Dr. Finkelstein's mortality study, but we seem to have in the Scarborough plant case, I gather, six mesotheliomas where the interval...though I'm not sure if it's the interval from first employment...well, no, it's simply

DR. DUPRE: (cont'd.) where the period of employment is less than nine years (sic).

That's different, of course, from his statistics on interval from first employment to death, isn't it?

THE WITNESS: Yes. And I...

DR. DUPRE: So we can't really substitute a figure for your zero there without looking at the data.

THE WITNESS: Actually, of course, there isn't very much comparison one can make between these two, because this is not a cohort study. This is just having ascertained all the cases of mesothelioma that I could find in Quebec, just allocating them to where they got their exposure.

Now, I'm afraid I don't know whether Dr. Becklake provided you with any information about that plant, about mortality, but I understand it was a very limited mortality cohort that was studied.

MR. LASKIN: My recollection is that we only dealt with the morbidity, and that as you indicated there was a doctoral student looking at the question of mortality.

THE WITNESS: Yes.

DR. DUPRE: Maybe one last question along these comparison lines for the Dr. Finkelstein study, counsel?

I was also very interested in your tab three, at page 436, where in table twelve which runs on for two pages, what you are reporting from different studies is lung cancers as a percentage of death, mesothelioma as a percentage of deaths.

Just looking at that last column, mesothelioma as a percentage of death, of course Dr. Finkelstein's outcome is way higher than any percentage that appears in this table.

It's around eighteen percent or something of that sort.

But again, from your knowledge would it be fair

DR. DUPRE: (cont'd.) to say that one reason why one perhaps wouldn't want to simply display Dr. Finkelstein's study in comparison with these others is that his cohort is so much more tightly defined as requiring nine years of employment or more, so on and so forth?

Or are there studies here with a cohort basically involving employees with a minimum employment period that is fairly substantial?

THE WITNESS: No, as far as I can remember none of these studies were based on a minimum employment period. They were all based on, as they should be, a certain minimum period between first exposure and follow up.

I would just like to say one more thing about Dr. Finkelstein's study. I mean, I think he has done an excellent study, but what I'm saying is that because you've got this very large proportion of mesotheliomas, and because you've got a mixed type of exposure, you don't know what the crocidolite exposure was compared with the chrysotile exposure, you can't draw any useful general conclusions about exposure response.

MR. LASKIN: It might be a convenient time to break for lunch.

Oh, sorry, Dr. Uffen.

DR. UFFEN: No, I just want to make sure I've got one thing clear. It related to...it's back in tab eleven where we were talking about table two and factory C, started in 1948?

THE WITNESS: Yes.

DR. UFFEN: The rest of the table there where the interval from first employment to death, it just can't be filled in yet, I guess, and presumably the number of cases may go zero, one, seven...

THE WITNESS: Twenty, thirty.

DR. UFFEN: Have you any comment about the implications of that?

THE WITNESS: No, it would be very unwise to speculate. We just have data on people exposed for that length of...we know that there have been people who worked from 1948, for that length of time since first exposure, but we can't...

DR. UFFEN: So to put it another way then, if we wanted to compare the results for factory C with A and B, we might have to reserve judgement for a little while, until we know what happened to the thirty to fifty interval years - interval from first employment?

THE WITNESS: Well, as I said, this is not... these are not cohort studies. That's to say they are not studies of these defined work forces, following them forwards. They are done the other way around. They are done by identifying cases of mesothelioma and looking at the pattern of their employment and finding which ones were employed in these factories, and then these aren't the periods since first exposure of these people entered in this table. This is just...no, I think perhaps they are, sorry. Yes, they are the individuals from when they worked first in that job, but just to look at the pattern of occurrence of mesothelioma in Quebec in relation to mining and milling and factory work, to see what you can get from that.

MR. LASKIN: Q. It doesn't tell us anything about how many persons were employed or exposed over time, or for how long...other than the actual cases...

THE WITNESS: A. No, that's right.

Q. ...which were shown on page 677.

A. That's right.

DR. UFFEN: It wouldn't be incompatible. That's all you can say at this stage...

THE WITNESS: Yes.

DR. UFFEN: ...except there may be quite a few more cases yet to appear.

THE WITNESS: Yes. And also we can't tell with these other factories, for instance, what proportion of all deaths that these mesotheliomas were. It could have been very high, or they could have been low. We just were not able to make the comparison at all.

DR. DUPRE: Can I ask you this, Dr. McDonald, going back to tab three on table twelve?

This paper reports that the table here was written before Dr. Dement's study.

THE WITNESS: Yes.

DR. DUPRE: Would Dr. Dement's study be a cohort study that could be filled out on that paper?

THE WITNESS: Yes, certainly.

DR. DUPRE: At the moment - I think you've already answered this, but I want to make sure that I have it clear - at the moment the Finkelstein study does not qualify because it does not look at the total number of employees during the period involved?

THE WITNESS: Oh, no, it does qualify in that if you take into consideration that you are only looking at people who worked for nine years, and that you've missed out the people who worked for shorter periods of time.

Certainly you wouldn't make a direct comparison, but you could see how the picture, the proportion of mesotheliomas fitted in.

DR. DUPRE: But the reason why you wouldn't make a direct comparison is because the other studies of this type have a cohort that is defined in terms of any period of employment, is that correct?

THE WITNESS: That's not entirely true, you see,

THE WITNESS: (cont'd.) because actually the...oh, yes, we haven't got...take Nicholson's study in Thetford Mines - but it's not entered in here, that again was later than this - his study might well be put into this context, and he took people who had only worked twenty or more years.

I mean, there are...I think some of these studies are probably people who have worked for some longer periods of time, but in general they are all people who worked for varying lengths of time.

I would say that you would have to make comparisons between these proportions of mesothelioma with considerable caution because the studies are not very comparable in a lot of respects.

All it was put together for was to examine what were the sort of order of events in different circumstances.

MR. LASKIN: Q. I guess if you put Dr. Dement's study in table twelve, just looking at his table seven which is in our exhibit four, he has a hundred and sixty deaths from all causes, and twenty-six lung cancer deaths, and one mesothelioma.

It would appear that his lung cancer percentage is roughly compatible with certainly the insulator and Rochdale textile operations. Of course his mesothelioma percentage would be very small.

THE WITNESS: It would be away down almost where the chrysotile miners are on that table.

Actually, that mixed products factory in Cardiff I think that was mainly, for most of the time, chrysotile, but there was a short period of crocidolite exposure, but that has a very low mesothelioma proportion too.

Q. As apparently does the...I suppose the other side of the coin is that you can also point to some other mixed fiber environments, and I'm just looking at the Paterson, New Jersey plant, which is amosite insulation, and yet the mesothelioma

Q. (cont'd.) percentage is low.

A. That's certainly true.

Q. I suppose if you look are enough...

A. You'll find all different...

Q. ...you can find a study to support your point of view.

A. That's very true, but you do, if you look at the report of the amosite insulation workers, you do find that they were a very odd cohort and they were very much older work force because they were employed during the war. They were the people who weren't conscripted for the armed forces and part of that explanation could be the fact of their age.

That is to say, they died of something else before they got their mesothelioma.

Q. Exactly. And actually I was going to pursue that question with you with respect to the Charleston study.

A. Yes.

Q. To what extent is there any possibility, in view of apparently large excess lung cancer, that these persons were dying of asbestos-exposed lung cancer before they had an opportunity to contact mesothelioma?

A. Well, it's usually the other way around. The mesothelioma deaths occur at a younger average age than lung cancer, and if you look at the numbers of deaths from lung cancer, that also would suggest that there is not very much possibility for this competing risk phenomenon, which is if people are killed off by accidents or something, in an industry, you might fail to find...if it were those people that were later going to get mesothelioma...you might fail to find the mesotheliomas.

But the mortality in most of these studies is not big enough to account for more than a very slight difference in the occurrence of cases.

Q. There isn't any of the age phenomenon in respect of the Charleston plant, that you mentioned with respect to Paterson, New Jersey?

A. Not at all. It was a very...it was a factory which employed local people mostly, whereas the Paterson plant didn't.

MR. LASKIN: Perhaps we should let Dr. McDonald have some sustenance.

DR. DUPRE: Shall we take our luncheon break counsel, until, shall we say two-thirty?

Or how is your outlook?

MR. LASKIN: My friends behind me will be pleased to know that I don't have many more questions to ask Dr. McDonald. She may be pleased, too.

DR. DUPRE: Okay.

MR. LASKIN: I don't know...perhaps I could just...

(REPORTER'S NOTE: Some discussion here among counsel regarding time element.)

DR. DUPRE: We will rise then until two-thirty.

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THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Ready to go, counsel?

MR. LASKIN: Ready to go.

DR. DUPRE: Proceed, please.

MR. LASKIN: Q. Dr. McDonald, is there anything further that you wish to say about either of Dr. Finkelstein's papers?

THE WITNESS: A. I don't think so. No.

Q. Could I just ask you one final question that really relates to a comment you made before lunch in relation to Dement's paper, and indeed your own study of Charleston, and that was...and I think your word was perplexing, the quantitative data on past exposures which appeared to you at least to be, as I took it, relatively low as compared with some of the, I guess oral evidence and pictorial evidence that you have both heard and seen.

One of the things Dr. Finkelstein did, as you may recall, in his mortality study, was took into account that his exposure estimates may be out by something of the order of magnitude of a factor of three or five. I'm just wondering whether that kind of approach might be usefully applied to the Dement paper?

A. Yes, I think that this is...since in epidemiology you are only getting the closest approximation you can, I think it's a very good thing to try to look at the likely range, the likely effects on your results, of being out by factors of one, two or three either way.

I would agree...it's very useful.

If you apply that to the Dement paper - we've already done this - and its factors are very much more than that, but the differences that, in which the results differ from say the miners.

Q. you have in fact done that?

A. Well, looked roughly at...they are only in an overall way...looked at what is the extent of the difference at a given exposure level, and that couldn't be accounted for by a difference of a factor of three.

It's more like a factor of ten or twenty, or more than that.

Q. I take it if one drew one general conclusion about Dement's paper in terms of all of the various comments or criticisms which have been made against it, would I be putting your judgement fairly to say that some or all of these may account for part of the difference or part of the startling result that Dement apparently has, but none of them really account for all of it?

A. I would agree with that, and I would agree that in that situation I think we have to accept that this may be the truth, and then we have to look for why this is and what should be done about it.

Q. Is there anything further that you want to add on the 'why it is' part?

A. There I can only speculate that either the state of the fibers as a result of the textile process have a much greater biological effect, or that some other possible exposure, potentiating agent, is present in that industrial setting.

I mean, there also remains the possibility of whether the shipyard experience of some of the men might have made a difference, but I really doubt whether that's important.

Q. We have talked about the question of fiber type in terms of mesothelioma risk, and I wonder if we might address the same question, but in terms of lung cancer risk, and I wonder if you could give us the benefit of your judgement as to how you see the epidemiological evidence in terms of relative risks of lung cancer, and the effects of different fiber types?

A. Well, we suffer from a terrible lack of exposure data, but all we can do is look in a very sort of gross way at differences between exposure in different situations. It looks to me as though when you have amphibole exposures that the risk of lung cancer is probably magnified by a factor of, perhaps two or even three, but it may well be that it's because the exposure is greater because of the dustier nature and finer division

A. (cont'd.) of the amphibole fibers.

So, I mean, there appears to be a difference as far as the risk of lung cancer is concerned, in that it's greater relatively with the amphiboles, but it's nothing like the order of the difference that exists of mesothelioma.

Q. When you make that judgement, can you tell us what particular studies you are thinking of?

A. The study by Hobbs in the crocidolite mines of Australia showed a risk, which was a mining exposure, showed a risk of lung cancer considerably higher than we observed in the chrysotile miners in Quebec.

Then when you compare the risks of crocidolite... well, it's a very rough measure as in the gas mask cohort we discussed earlier. It looks as though the lung cancer risk was perhaps very roughly double...or at least proportional mortality was double that you got in chrysotile exposure.

I can't remember...I think there are one or two other studies in which the...but I'm afraid I haven't got them on the tip of my tongue, or in my mind.

DR. DUPRE: Is table twelve, again at tab three, at all useful here? Because on page 437, lung cancer deaths as a percentage of all deaths for chrysotile mining and milling in Quebec and in northern Italy certainly do appear to be substantially lower than in the insulation factory workers.

THE WITNESS: Yes. But of course we don't...these populations aren't necessarily comparable. It's just that there is a higher proportion of mortality in the insulation cohort.

That is compatible with a higher risk in crocidolite...well, in amphibole exposure. But of course it isn't much greater than the Dement...do you have the Dement proportional mortality for lung cancer? I don't have his paper in front of me.

MR. LASKIN: I suppose we might also...in terms of

MR. LASKIN: Q. (cont'd.) relative risk exposure your own table at tab thirteen, your Cold Springs Harbour paper, is of some help in relation to all the studies.

THE WITNESS: A. Yes.

Well, we don't have in this paper the proportional... in this table the proportional mortality. It doesn't really help very much for that.

Q. But in terms of...I'm just thinking of Hobbs' study in this table, and I'm...I don't know whether it's me or the typing...but is the observed to the expected thirty-eight to twenty-two point six or thirty-two point six?

A. I think it's...oh, it looks in my paper like twenty-two point six.

Q. It may be that...

A. No, it must be thirty-two point six...

Q. Because they...

A. ...because of the difference...five point four.

No, it isn't clear, as you say.

It could have even been a typographical error...

Q. It may be.

A. ...it certainly looks like a two there, but it clearly ought to be a three.

But he has a proportional mortality of about twelve for respiratory cancer, which is about double what you would expect in a general population, and is therefore not very different from what I was saying that occurred in the amphibole-exposed people.

Q. I suppose it leaves me to ask whether in your view, in terms of lung cancer risk, whether perhaps it's the process or that part of the asbestos industry which may be a more important factor than the differing fiber types?

A. Yes, I think that there is clearly a difference

A. (cont'd.) between mining and processing in chrysotile, but there is, nevertheless, a substantial risk of lung cancer in mining chrysotile, and just the difference between the different fiber types as far as lung cancer is concerned, is nothing like so great as it is for the amphiboles....that there are differences, but they aren't of an enormously large order.

Q. Can I just briefly go back to one issue we dealt with before lunch, and that was the papers that look at lung tissue, they analyze lung tissue, and it might be helpful if we...I want to make sure we all understand the tables that you've presented in respect of those papers, and perhaps we can go to tab fifteen, table number one at the back.

Table fifteen, I take it, is the analysis that was done from the mesothelioma series in North America, as at 1972?

A. Yes. Well, it was...yes, it was as of 1972, in both the United States and Canada.

Q. The mesotheliomas that you studied, were they...was there any premeditation in what you selected, or was it simply whatever you could get lung tissue for?

A. They had to be from autopsy cases, otherwise you couldn't get a specimen of lung tissue. I took all the autopsied cases and requested from the pathologists a piece of lung tissue for both the case and the matched control.

This series that was studied was every case and matched control that I was able to get out of that series. I think there was something like a hundred and seventy-two autopsies, with cases and matched controls, and we've got ninety-nine of them.

But they weren't selected. They were just whatever we could get.

Q. To come back to a question Dr. Mustard asked this morning, was there any premeditation in terms of what part of

Q. (cont'd.) the lung the specimen came from?

A. It wasn't possible to do anything about this. We would have liked to have specimens from defined representative areas, because there may well be differences as much as..according to Pooley...as much as twofold in the asbestos content of one part of the lung compared with another, but these were just whatever specimens that pathologist was able to send, and they could be from any part of the lung - for the case and for the control.

Often where there was a known respiratory problem, they often took a number of specimens. But sometimes they only kept one for some of the autopsies, and that was it.

Q. And for the purpose of this table, I take it, the fiber is defined simply as having an aspect ratio of three-to-one or greater?

A. That's correct.

Q. Can you help a layman here and tell me what per G dried lung is?

A. Gram of dried lung.

Q. What are we looking at on this table? Can you just explain to us what we are seeing on this table?

A. What was done was, the series of ninety-nine... well, say we take the male cases, I think that was seventy-six - yes, seventy-six male case control pairs...the pieces of lung tissue were taken and the control's, and they were all jumbled up and sent in a new numerical order to Dr. Pooley, who, by a process of dry ashing and use of sodium hydrochloride, or some other substance, dissolved away all the organic tissue and then having... or having taken the dried lung, I'm sorry, first of all, and weighed it, and then finished off by removing all the organic tissue, he examined parts of this on the grid under a transmission electron microscope with an x-ray energy dispersive analyzer, so he counted say a hundred...I think it was generally a hundred fibers and estimated from the area that he had how much...what weight

A. (cont'd.) this was derived from, and was able to express these fibers as millions of fibers per gram of the dried lung tissue that he examined.

Q. Of each type of fiber?

A. Of each type of fiber.

Q. In respect of the cases, because I take it the cases came from the two hundred and seventy-four, whatever the number was, of mesotheliomas in North America.

Were there, amongst the cases that formed part of his case control series, were there mesotheliomas for which there was no prior known asbestos exposure?

A. Oh, yes. Over fifty percent...fifty percent would have had no known asbestos exposure, in the males.

Q. In the males.

Was there any magic to the categories that you've chosen down the lefthand side of table one - in less than one, ten to the sixth, and one to ten, and so on?

A. No. It was...obviously these are...you should always define your categories in some sort of way and not fiddle the answers according to what...fiddle categories according to the way the answers are coming out, but it was that...that looking at the numbers of cases that had these various amounts, the table is presented in this way, although the actual analysis was done in a larger number of groups.

This is a condensation, but done from sort of first principles on the numbers of fibers and not according to the way the answers came.

DR. UFFEN: Can I ask a few questions here, because I wanted to clarify something?

THE WITNESS: Yes.

DR. UFFEN: We're quite taken with your colleage, Dr. Gibbs', reference to the discovery of significant

DR. UFFEN: (cont'd.) amounts of tremolite in the lung tissue of your miners. This is your very recent draft paper and the last one, number sixteen.

THE WITNESS: Yes.

DR. UFFEN: I can tell you what my concern is right at the moment. My understanding is that tremolite occurs in other kinds of rocks as an impurity. It occurs in limestone, which is a very common rock used in road building, construction and so on, so that if tremolite turns out to be significant we might have a vastly different control population than we would have been talking about so far.

THE WITNESS: True.

DR. UFFEN: So I would just like to explore is, the importance or otherwise of tremolite with respect to mesothelioma, lung cancer and fibrosis of the lungs.

Now, in table one that would...that I'm talking about...I see that the amosite, crocidolite and anthophyllite, the cases are roughly three times the controls, in the total numbers, which appears to be quite significant.

But the tremolite is very similar to the chrysotile in that the cases and controls are very similar.

The first thing that strikes me is, here we have an amphibole - namely tremolite - which is similar to the serpentine - namely the chrysotile - even though their chemical structure is quite different.

So this sort of points to a non-chemical explanation, maybe the physical size.

But then this leads me to the question: In your last paper where you were talking about the identification of these in the lung, and you used the transmission electron microscope method...on page four in particular...it talks about the aspect ratio of three-to-one or greater, but it doesn't mention

DR. UFFEN: (cont'd.) the length.

Was there any restriction on the length of the fibers?

THE WITNESS: No, there wasn't. We would have liked to have put one. In fact, I think probably we do have it broken down by less than five mu and more than five mu, but we haven't got it broken down more than that. It just wasn't feasible for Dr. Rowlands to do this in the time available.

DR. UFFEN: But these data that are presented in tab sixteen, ma'am, include all the fibers - even the little ones - that the membrane filter method doesn't measure?

THE WITNESS: Yes. That the membrane filter...?

DR. UFFEN: You are using a transmission electron microscope?

THE WITNESS: Yes.

DR. UFFEN: Which is able to measure much smaller fibers?

THE WITNESS: Yes.

DR. UFFEN: They may still be three-to-one aspect ratio, but there may be a whole lot of little ones in there....

THE WITNESS: Yes.

DR. UFFEN: ...that you wouldn't measure if you were doing the membrane filter method. So the data presented in tabs fifteen and sixteen...

THE WITNESS: All right. Could you clarify 'what you wouldn't get in the membrane filter method'?

DR. UFFEN: They don't count...

THE WITNESS: They don't...

DR. UFFEN: You can't count them...

THE WITNESS: But you can count them by an electron microscope with energy...and idenfity them by an energy

THE WITNESS: (cont'd.) dispersive analyzer, in just the same way as you can in tissue.

DR. UFFEN: Yes, you can identify them, but most of the information that has been presented to us up to now, and certainly by Dr. Gibbs when he was here, was from...

THE WITNESS: Optical microscopy.

DR. UFFEN: ...optical microscopy..

THE WITNESS: And phase contrast.

DR. UFFEN: ...results taken from a membrane filter measurement?

THE WITNESS: Yes, that's true.

DR. UFFEN: Would I be safe in concluding then that there is a possibility that some of the results in your most recent papers, tabs fifteen and sixteen, could be attributed to small particles, not measureable in the normal optical microscope MFM method?

THE WITNESS: Oh, yes. Because I think that the optical microscope just measures a proportion...measures fibers longer than five mu...and that is generally taken to be an index of the whole range of fiber sizes.

DR. UFFEN: It may be fairly obvious and simple to you, but I'm still trying to sort out the importance of small fibers and the difference between their physical size and their chemical properties and so on.

Could we turn now to the fibrosis of the lung, the asbestosis? I was struck by the final comment in your tab sixteen, on page six:

> "Neither this investigation nor the earlier study by Pooley were designed to evaluate the extent to which tremolite contributed to the pulmonary fibrosis of chrysotile miners and

DR. UFFEN: (cont'd.) "millers, or indeed to others.

This clearly deserves to be done, but will require comparison of fibrotic and other changes in chrysotile workers exposed to varied quantities of tremolite".

Is this something that we should take very seriously from the point of view of a Royal Commission trying to make recommendations for the future, or is it merely of a minor nature that requires some research to tidy it up?

THE WITNESS: I suppose I'm speaking with a certain amount of ignorance, but I would have thought that as far as mining in Quebec is concerned, where there is quite a lot of tremolite in portions of the rock - not in all of it, I gather - what tremolite does is of considerable importance. You know, we do need to find it out as far as chrysotile mining is concerned, but I think that by the time the chrysotile from Quebec gets to the manufacturer, I don't think there is much tremolite left in it...but I am not sure about that.

DR. UFFEN: I was thinking about ultimate regulations that specify, perhaps, so many fibers per liter that mustn't be exceeded, that we might subsequently discover that there is tremolite in many other walks of life to which this regulation ought to apply, and never dreamed of it at the time we were thinking about it.

For example, if we go back to what is getting to be an important table - twelve, in tab three - the very last item is talc, including tremolite, in New York State, where nine point nine percent of the lung cancers - nine out of ninety-one - presumably were due to the tremolite.

That's not anywhere near as low as the chrysotile, but it's about halfway to the crocidolite and amosite.

DR. UFFEN: (cont'd.) You know, should we be worrying about talc? Babies' bums, you know, generations have been powdered and patted...

THE WITNESS: Well, I suppose that comes in with the whole area of lower exposures to different sorts of asbestos, of the population. But industrially it appears to be a rather limited phenomena...unless I'm wrong and there is a lot of tremolite that gets processed with the chrysotile.

DR. UFFEN: Just one more thing to see whether... we've had the evidence before us that mesothelioma increases with time after exposure at a rate which goes up as a three-and-a-half caliber of time. I'm thinking about children again. That's a smaller amount than you would ever consider dangerous for a worker aged thirty. For a child aged three or three months, exposed to talc, are we in any position to make prognostications about what will happen thirty years later?

THE WITNESS: Well, we have some limited information on exposures to presumably workers' dusty clothing in the home, to children in the home, and we have a few cases of mesotheliomas which appear to arise from this, which occur in the thirties with a similar sort of latent period. But we have too few to really make that a firm statement.

My feeling about talc is that as with chrysotile in the atmosphere, it's a problem but probably the dose that is received is very unlikely to be very substantial because it's only...the child might have a little cloud of talc as it was being put on, but it doesn't last for more than a moment.

The whole question of tremolite, I think, does need to be investigated. But I think that possibly that has a lower priority than some of the other gaps in our knowledge.

The question of tremolite and fibrosis was

THE WITNESS: (cont'd.) suggested by Pooley when he examined the miners, the lungs of chrysotile miners, but there is really nothing to support it.

But since it's there, it persists. It should be considered as to whether that contributes to the fibrosis.

DR. UFFEN: One last question. In the identification of the lung tissue, has anybody ever identified any actinolite, which is the one leftover amphibole we haven't heard about yet?

THE WITNESS: Now, Pooley did find a little actinolite in one or two cases, but it wasn't enough to...I don't remember how many it was. I didn't bring the original data with me here, but certainly he did look at that, and there was very little.

He looked at also...not just the asbestos fibers... but he looked at the whole range of mineral fibers and identified everything that he could, and there doesn't appear to be anything else, except the asbestos fibers, that show a difference between cases and controls.

MR. LASKIN: Q. During that discussion with Dr. Uffen, he did mention some evidence which we indeed have had in relation to mesothelioma, that unlike lung cancer it is independent of age. I am wondering whether your own research on mesothelioma would lead you to support that view or not?

THE WITNESS: A. When you say independent of age, what do you mean?

Q. That the evidence we have heard is that in relation to lung cancer one should be looking at the background rate of lung cancer in the population, and you don't see...you are unlikely to see asbestos-exposed lung cancer of people who are younger because there just isn't much lung cancer in the population. Whereas the older people get, the higher the

Q. (cont'd.) background rate and that's when we see a lot of lung cancers.

Mesothelioma, we are told on the other hand, you ought not to be directing your attention to that question, but rather the time since first exposure, it being more a function of time to some power.

A. Yes.

Q. Does your own research support that view?

A. I would say so, yes. There isn't any...well, I don't know that we have the numbers, but it looks as though it is a function of the interval. But for children, I suppose one always suspects that children might be at higher risk perhaps, because of their state of their growth, but otherwise for other people there doesn't appear to be...there appears to be a fairly standard interval between first exposure and death from the tumor.

Q. The only other...thanks, Dr. McDonald.

The only other topic I wanted to ask you about in a way follows on from Dr. Uffen's discussion with you, and it's the paper that appears at tab five of your compendium, which is the study that took you to the Homestead Gold Mine in South Dakota.

It has been an issue that has concerned the Commission as to whether we have to worry about asbestiform exposure, exposure from asbestiform in other than strictly asbestos mines - particularly bearing in mind our own situation in Ontario where we have really no operating asbestos mines, but lots of gold mines and iron ore mines and so on.

One of the things that we have been trying to do is grapple with this study as opposed to the study that was done by the NIOSH group, which apparently produced some...came to a different conclusion.

When Dr. Lemen was here and gave evidence before

Q. (cont'd.) this Commission, he commented on the study that appears at tab five, of which you were a coauthor, and as I recollect his evidence, one of the things he suggested was that you were rather more looking at a survivor population because of the cohort that you selected, and therefore perhaps, I take it, were unlikely to see any excess mortality if indeed there was some.

Do you have comment on that?

A. Yes, I have two comments.

One is that in the paper by Wagoner, et al, there were inconsistencies. At least the risk of lung cancer was higher in...I can't remember if it was people who...I think it was the interval between exposure and developing the lung cancer. It was higher, the incidence was higher, or the proportion of the mortality was higher, and the shorter...after a shorter interval than it was after a longer interval.

So that's an inconsistency. And the second thing is that....you are going to get clarification on this, aren't you, by the results of the Stanford research groups' re-examination, new study, of this population? Have you had any information on that?

I don't think it's published yet.

Q. My recollection is that one or two witnesses may have made some reference to some work being done in California, and I take it that is it?

A. That is it, yes. It was commissioned by NIOSH and the Stanford research group have undertaken a large scale study, and I don't know whether it's published so I don't know whether it would be in order to comment on it.

I can only say that I think that it will resolve your questions, although there is still a little bit of lacking information. But on the whole...and NIOSH is at the minute

A. (cont'd.) trying to get some information filled in, I think.

Q. Do you have any knowledge, or are you able to help us, on what it demonstrates?

A. As I say, I don't know that it's in order for me to talk about that report. If you tell me that it is, I will. But if you don't, I don't think I should.

Q. Well, I...short of knowing under what circumstances you are privy to the document, I...

A. Well, I can only say we are quite happy about the Stanford report as it stands at present. But, you know, it isn't final, so obviously we can't...

DR. UFFEN: Were you asked to referee it as...

THE WITNESS: My husband has got it at the moment for refereeing.

DR. UFFEN: I see. That is awkward if you are supposed to be refereeing.

THE WITNESS: Is that public, though?

DR. UFFEN: It's certainly awkward.

THE WITNESS: Oh, it's awkward. Oh, yes.

MR. LASKIN: Well, that's fair enough.

I have no more questions, Dr. McDonald. Thank you very much for your patience.

My friends here in the back row may have some questions for you.

THE WITNESS: Thank you.

They probably won't be so kind to me.

MR. LASKIN: Oh, they are very kind.

DR. DUPRE: Thank you, counsel.

Have I got a batting order?

Mr. Hardy?

MR. HARDY: No, I'm not first today.

DR. DUPRE: Mr. McNamee?

MR. McNAMEE: Yes.

CROSS-EXAMINATION BY MR. McNAMEE

Q. I have a few questions, doctor, primarily in relation to your ongoing study on the same factory that Dement studied, and Mr. Laskin asked you some questions, and if I repeat any questions he asked, please forgive me.

I think one of the criticisms by Mr. Peto of Dr. Dement's study was that he moved the deaths into the very last group and created an inordinate standard mortality ratio of observed-against-expected deaths. He had different categories of deaths, and I think all the deaths were moved into one group.

Did you consider that an appropriate technique?

A. Well, I think Mr. Laskin asked me about this.

Q. I'm sorry.

A. I was unaware that that was the case, and when we had a group which included a number of statisticians, which was trying to look at the Dement results and see how... because they appear to be very important...to see whether there appeared to be anything wrong, this wasn't pointed out. But Julian Peto is a very clever statistician and maybe cleverer than the ones who had looked at it earlier.

I'm not aware that that's the way the analysis was done.

MR. LASKIN: I think in fairness to Mr. Peto we should perhaps clarify the record by saying I think it was Dr. Finkelstein who actually...

THE WITNESS: Who made that statement.

MR. LASKIN: Who made that statement.

MR. McNAMEE: Q. In addition, Dr. Dement's study does acknowledge that smoking played a role in the lung cancer, but he then goes on to say that he can't account for all the deaths by

Q. (cont'd.) attributing them to smoking.

However, reading the study, he really didn't seem to make any attempt to factor out what percentage of deaths might be properly credited to smoking...at least I didn't think so.

What was your opinion of the analysis of the smoking effect in comparison to lung cancer...or insofar as it affected the lung cancer?

THE WITNESS: A. Well, I think he showed that the smoking habits of the population didn't differ from the reference population in the United States, but he didn't have any smoking data on the different exposure groups, and it would only matter if the different exposure groups had different exposure patterns...if they all had similar smoking patterns, I'm sorry, it wouldn't affect the slope of the line.

So I would say that we can't be sure that smoking hasn't acted as a confounding variable in this, but I don't see any reason why it has.

Q. Dr. Dement didn't have any in depth analysis of the occupational experience of his cohort prior to the time they commenced employment in that factory. Will your study attempt, at least, to have some kind of analysis of the prior occupational exposures of your cohort?

A. Well, that brings up the question of how I'm going to try to look at the question of the possible naval yard employment, and I think since one of my analyses is going to be case-control analysis, taking all the respiratory cancers and taking a series of matched controls, I would like at that point to investigate in detail the possibilities of earlier exposure in both these groups, but it would have to be done in a blind fashion with the whole group of cases and controls, investigating systematically what their other employment history was.

Q. Also, his control group was basically, I think,

Q. (cont'd.) the United States population in general, and one of the previous witnesses indicated that he might have also used the local, either the local state death rates or even the county in which this plant was located.

Is your control group going to be based on the United States standard population, or something more specific?

A. My reference group, my population, reference population, is the State of South Carolina, but in fact I don't think it's likely to make any difference compared with Dement's results because the death rates from respiratory cancer appear to be rather similar in South Carolina to the United States in general.

Q. I think one witness indicated that the lung cancer rate in that particular county was almost twice the national average?

A. That's true for the county, but not for the state.

Q. Would that county rate be unduly influenced by this rather large group that worked in the textile mill?

A. I think that the county rate could be...I don't know that the factory would influence it very badly. It could influence it, but one would need to see what the population sizes were who worked in the factory, and then the total county.

But I think that the naval shipyards in the county would...in which the employment...the numbers of persons employed were very much greater than in this plant, that would affect the county rate, certainly.

Q. Now, Dr. Dement had a number of conversions there. Will your study...is your study going to attempt a similar conversion, or are you going to work it on particles and then... particles for one group and fibers...

A. Well, I'm going to go out and seek guidance

A. (cont'd.) from different people on what could be done about this conversion, and I plan, as was suggested earlier, that one looked at the whole range of possibilities of orders of difference, then one could present that analysis using these different fiber conversions.

Q. Would you have any objection to just using the same conversions that Dr. Dement suggests? Have your experts reviewed them and...

A. Well, I would rather get some set...you see, I would like to analyse mine, in the first place, in the measurements in which the exposures were taken - in million particles per cubic foot - because those are available right up to 1970, and there they cover the whole period that's relevant to the causation of lung cancer.

But after that, I really haven't decided how to face the rest. As I say, I would really like to go to a group of environmental people and say 'what do you advise me to do about this'...about what is the best conversion factor to use for the various jobs in this factory.

Q. One final question with respect to Dr. Finkelstein's study. Do I understand that one of your criticisms is that this represents just a small slice of the actual working population, and that the heavily-exposed portion of the working population? Whereas, say in comparison to your husband's studies of the Quebec miners, he took...at least he went through every one of them and then divided them into...finally picked his cohort out of I don't know how many thousand workers it was.

And to just give you an indication, I understand Johns-Manville had perhaps five thousand people working between 1948 and 1981. That's just a figure that has been given to me, I can't vouch for it, but perhaps five thousand people have had asbestos exposure at one time or another at Johns-Manville.

A. Definitely I would say that you stand a much better chance of getting the most, the best exposure-response information if you look at a complete cohort, including shorter-term employees and lighter exposures, because then you've got a bigger range of exposure to get your dose-response curve out of.

But on the other hand, as I said earlier, having done that, this is a real problem because of the unknown relative amounts of crocidolite and chrysotile, so that you are only able to calculate your response to some unknown mixture of two different fiber types, and therefore this isn't very generally useful.

MR. McNAMEE: Thank you, Doctor. Those are my questions.

DR. DUPRE: M. Casgrain?

CROSS-EXAMINATION BY M. CASGRAIN

Q. Dr. McDonald, I gather from what you have stated in connection with Dr. Dement's statement, one of the things that you propose to do in your further study is to classify the subjects by exposure.

I wonder whether you could help me a little bit along these lines. Are you referring to...are you saying that you will attempt to do with those workers approximately the same thing that you have done with the workers in the Quebec mines? That is, find out exactly what their own occupation entailed by way of exposure, is that what you mean?

A. That's correct.

Q. Now, is there enough data in Dr. Dement's... supporting data, shall we say...to permit you to do that?

A. I am not, first of all, taking it from Dr. Dement. I already had a hygienist, a lecturer in hygiene, visit the factory and collect all the exposure data that there is, and so I'm starting from the same point at which Dement started, and

A. (cont'd.) I'm, at the minute, working on all of the work histories, and for each department and if possible for each job within each department, this environmental expert is going to give me his best estimate, from the data that he has, of the exposure that that person would have received in that time, which is the same as we did for the miners except for the miners we had to do a certain amount of backwards extrapolation, and here we do start with some data in 1930, and there are very few men...there are a few who go back earlier...and we shall just have to extrapolate those backwards.

Q. Do I understand that this was not done in the case of Dr. Dement?

A. Yes, this is what he did.

Q. You are doing the same thing?

A. Yes. He estimated...

Q. With your own personnel?

A. Yes.

Q. It may sound like an obvious question to you, but why would you repeat what he has done?

A. Well, first of all, I have a different cohort, I have a bigger cohort, and they worked in different jobs. It doesn't make the job any easier, that he has done it, if I have got a different set of work histories.

Well, I have anyway to define the jobs and the times in which all of these employees worked, and then having got that we then have to match up the exposures that...the best exposure data that we can for that job and that time, but taken from the same thing as Dement has, and it's just as easy to start from scratch as it is to start with what Dement has.

Q. I would have thought an update of Dement would have been sufficient?

A. No, but it would be more complicated, the way he has got it, the form that he has got it in.

Q. Is it possible, perhaps, that, depending on the technique you use to cause this evaluation to be done, that the results that you would reach in respect of exposure, individual exposure, would be different from those that Dement reached? Is that possible?

A. Well, you mean apart from the errors involved in estimating individual's exposures? I mean there are some errors involved.

Q. Mmm-hmm.

A. I would have said it was much better for our state of knowledge that we did start from the basic data and do our own analysis, than use anything that he has already done.

Q. If only to double check or prove or disprove?

A. That's right. Well, I mean, I don't think it's any more difficult and I think it's the only way that the answers are going to be credible.

Q. I take it then that I can assume that the Dement study so far has not, in effect, applied these criteria to the conclusions of the study? That is, individual exposure?

Can I say that?

The Dement paper so far, has it in effect attempted to classify...

A. Yes.

Q. In a way satisfactory to you, the individual exposures?

A. Well, the way that I understand that he has done it, it seems all right. But I'm going to have to look at it again if Dr. Finkelstein says that this has the effect of classifying people, of putting their deaths into the highest-exposure-group.

I just don't understand that, I don't understand what is being suggested.

A. (cont'd.) But from the point of view of calculating the exposures for each individual so that in the analysis people can be grouped according to different exposure, total cumulative exposure, I shall probably do it the same as Dement has done it, as far as I know.

Q. Correct me if I'm wrong, but I envisage, for instance, that if you had on the specific floor of a factory an index of exposure of, say five fibers, it could be that someone working next to a carding apparatus would be more exposed than he who would come in to sweep the floor, or be a millwright and come in from time to time only. Correct?

A. Yes.

Q. That is the kind of classification you propose to do?

A. Yes. I think we are not going to be able to do a very fine classification. I think my environmental colleague is going to take each department, each location and then probably subdivide the people working as to those who would have received average exposure and those who got more, and those who got less, and in a rather arbitrary way. It's all rather... it's inevitably imprecise when you are estimating past exposures, but you see all that does in epidemiology is to confuse, is to mask findings rather than create them.

It tends to dilute results. But what you are looking for is differences between low exposures and high exposures, and you would tend to get a smaller difference if your...to the extent that your individual measurements may not reflect the truth.

Q. Well, if I may, just to try and make comparisons between, say the Quebec mines and the Dement study in respect of environment, and I have a table here...I think perhaps

Q. (cont'd.) I should show it to you first and then ask you questions on it so that we both understand.

It's table six in his study. I'll pass it down to you.

DR. DUPRE: Table six in Dement's study, M. Casgrain?

M. CASGRAIN: Yes.

Unfortunately, I haven't got my copy, so...

THE WITNESS: Well, maybe I should have brought mine. I've got one somewhere.

M. CASGRAIN: Q. Perhaps if I stay close to you, we can use the same one at the same time, and nobody will accuse me of crowding the witness, I hope.

I also need my glasses.

Now, if I look at table six, I look for instance at what is called here 'heavy weaving', and I look at what the exposure was between 1930 and 1936, and I see nine point two fibers per c.c. Right?

Now, could I perhaps try and compare a machine of that nature to, say, a cyclone vibrator in a mine? You know what a cyclone vibrator is in a mine?

THE WITNESS: A. More or less. I'm fairly ignorant about this side of it, so don't push my knowledge too far.

Q. Well, I'm talking about apparatus you would find in a mine, which would also create dust.

A. Yes.

Q. Or perhaps we could compare it to the bagging area at the time when there was no automation. Could we do that?

A. Mmm-hmm.

Q. Now, would you expect that heavy weaving

Q. (cont'd.) and bagging would perhaps cause the same amount of dust?

A. No, I think I would...well, it depends under what conditions the bagging was being done. You mean open bagging when...

Q. Well, open bagging to an extent. Doctor, I'm trying to find a comparison here, because if you look...

A. I don't think that you can easily make these comparisons, and I think I look at Dement's things and think, goodness me, those look all very low compared with what the case was in the mines. But I mean, I don't know how to interpret that.

Q. That's my reaction, too. That's why I'm looking at heavy weaving. Would you not say that heavy weaving, in effect, would create about the most dust in a factory?

A. Well, I don't know. I would say that the preparation and carding are very bad.

Q. If we look at carding, between 1930 and 1935, you had ten point eight fibers. Does that make any sense to you?

A. Well, this whole question was looked at by a group of people, among whom were people...Eyre was there from the American Public Health Service, who was actually involved in some of the measurements here in this plant, in the American Public Health...the U.S. Public Health Service surveys, and all that I can say is everybody seemed to accept that they didn't have any real reason to say that these measurements weren't correct, but we only concluded that if these measurements were correct, then it must be that the biological effect of the... of whatever...that what they were measuring was not what was having the effect - that the effect must be from something that wasn't being measured, perhaps.

Q. But you see, taking into account what you

Q. (cont'd.) have said and what your husband has said as well, in respect of cleavage of the fiber, and the fact that the fiber, when factored - if I can use the expression - when manufactured or going through that process cleaves into a great number of smaller fibers, and if I look at a figure of ten point eight fibers per c.c. in 1930 on a carding machine, where obviously the controls are far from being what they are now today, or even fifteen years ago, it seems to me that this figure is completely out of proportion.

Do you not agree with me? That those ten fibers should be a hundred fibers, judging from the evidence we've heard in respect to fiber cleaving as it goes through the factor? Would you not agree with that?

A. Well, I agree that at face value it appears like that, but I have no evidence to say that those measurements aren't correct.

But they must be measuring something different in the mines than the factory.

Q. Yet this was being done with the impinger method.

A. Yes.

Q. Albeit it was not a midget impinger...

A. No, they used a large.

Q. ...but a large impinger.

A. Yes.

Q. But the results would come out the same, would they not, on the impinger?

A. I understand they are supposed to.

I agree with you, but it just is insoluble and I can't throw any light on it.

Q. I'm looking, for instance, at heavy weaving and I see that between 1937 and 1975, it was two point six fibers.

Q. (cnt'd.) Should I assume that the number of fibers would be the same in 1937 as in 1975, although there would have been improvement in the equipment used to in effect absorb the fibers...not absorb, but to eliminate the fibers through air suction method, and what have you?

A. Well, as I understand it, this plant was supposed to be a good plant. It was supposed to be an example in dust control, I think in the late-thirties, to other textile plants.

Q. In the late-thirties?

A. And you also have to say that the textile process hasn't changed at all, until now they've got rid of this process, this dry process, and they have got now, at the Charleston plant...

Q. The wet process?

A. The wet process.

But, I mean, I think that the small change over the years is compatible with not changing the process.

Q. And using the same equipment for over thirty years?

A. Using the same equipment, that's right.

Q. So that...this perhaps is a very simple-minded question...so that if I had today to determine whether...if I am going to do heavy weaving in my plant, I should think of the Dement experience. Obviously I would not, because I would be using a wet process. Is that not correct?

A. Yes.

Q. So that in effect if I looked at this kind of data, having to do with a factory, and if I assume that most of the equipment is outdated and has in effect been changed to the wet process, it becomes somewhat difficult to use that data and apply it to a modern factory, does it not?

A. Yes.

(REPORTER'S NOTE: Slight pause in proceedings)

M. CASGRAIN: I'm sorry, Dr. McDonald. I was just trying to think up a very learned and intelligent question. It's a difficult one too, and my problem is I have to understand it before I put it to you, which makes it even more difficult... which is what happens when you try and examine experts.

Anyway, forgetting all the lessons I got in law school, I'm going to ask you the question anyway - because I don't know what the answer is going to be. Apparently one shouldn't ask those questions.

Was there not a lot of asbestosis in that plant as early back as 1930?

THE WITNESS: A. There was a report by Metropolitan Life, but I thought it was in the later 1930's.

Q. Yes?

A. Indicating a number of cases of asbestosis.

Q. Now that would be in the late 1930's, did you say?

A. I think so.

Q. Would that be compatible, asbestosis at that stage, in that plant?

A. With what?

Q. With asbestosis? With the kind of exposure that asbestos calls for?

A. You mean at those levels?

Q. Mmm-hmm.

A. Well, I mean, compatible on what basis? You wouldn't expect it, if you were dealing with the mining population, you wouldn't expect...well, it would partly depend on how long the people were exposed, but all the same that wouldn't seem a very high level of exposure and I suppose you wouldn't expect much. But we don't know what proportion of

A. (cont'd.) employed persons had asbestosis.

Q. Neither do we know what their individual exposures were?

A. No.

Q. And it could be, for instance, that of all the cases of asbestosis they were all people who were working on a continuous basis next to the heavy weaving equipment, as opposed to others who were ambulating throughout the plant? Is that correct?

A. Well, I'm afraid I can't remember the details of this study, but I think they did take all the people who had worked for a certain time, and I think it was irrespective of what they were working at, and they found a number of cases of asbestosis among them...

Q. Not knowing...

A. ...and I can't tell you...it wasn't a very precise study.

Q. And not being able to tell whether those very cases were people who had, in effect, worked on a continuous basis next to weaving equipment?

A. They didn't look at exposure - didn't examine or report on the exposure history.

Q. Because I take the case of a millwright in the mine, for instance, who will circulate on the floor but will only occasionally inspect equipment, his exposure is less than he who had worked next to a piece of equipment? That would be the same situation, wouldn't it?

A. Yes.

M. CASGRAIN: No other questions.

Thank you very much, Dr. McDonald.

DR. DUPRE: Miss Jolley?

MISS JOLLEY: I just have two questions, because

MISS JOLLEY: (cont'd.) both Dr. Uffen got to my question before on tremolite, and John covered a number.

CROSS-EXAMINATION BY MISS JOLLEY

Q. I would like to go back to the conversation you were having with John just before lunch time, and that was - we were dealing with the whole issue of competing causes of death and the possibility...we were talking about the Charleston study, and when John asked you if it was possible that perhaps some of that population...oh, and I think Dr. Mustard asked about that...if it was possible that some of the population may have died from other causes and therefore were not available to die from mesothelioma, and you said that, well, generally mesothelioma kills people at a younger age.

A. Than lung cancer.

Q. Right. But then we look at your study on mesothelioma in tab eleven, in Quebec, where we are discussing the latency with chrysotile, and you have people dying of mesothelioma much, much later in life when they are exposed to chrysotile than with crocidolite, and so I would wonder whether it isn't possible that they might have died of competing causes in the Charleston...

A. Well, if that was so, you are suggesting perhaps that there is a difference between the amphiboles and chrysotile, that chrysotile only causes pleural mesothelioma and twice as late in life as the amphiboles do, and you are asking me whether there could be competing causes of death in the Charleston factory in that people died of lung cancer before they got to the...

Q. To the latency...

A. ...had a chance of getting mesothelioma of the type that we appeared to get in the chrysotile miners.

It's perfectly possible, but it just doesn't

A. (cont'd.) seem to make very much sense to me that although there was a high mortality from lung cancer...what I would expect if there were a competing cause like that, that you would nevertheless get cases, you would be bound to get some cases of mesothelioma, but you might be getting less than you would have done because some of them might have died earlier from lung cancer, but it wouldn't prevent the occurrence. Competing causes of death never do that - they just influence the level.

Q. Right. I was just having trouble reconciling the long latency for chrysotile, and your comments.

A. Yes. You see, it wouldn't be one for one that each person who did get lung cancer, who was going to get mesothelioma, had died earlier of lung cancer. Some would have failed to die from lung cancer and would have survived to get their mesotheliomas.

Q. I have a question about your mesothelioma and fiber type, and it's a bizarre question, but I am interested in why did you decide to study those three plants?

A. Oh, for a very easily-explainable reason: Because in the chrysotile mines we had first of all relatively very much fewer cases of mesothelioma than we realized were occurring in insulators or in products factories which were all the factories that had been reported...were mixed factories, had amphiboles with them. So it was obviously very desirable to look at large and old factory populations which had manufactured only chrysotile.

The only two reports of studies prior to my starting this study were two small factories, one in America by Weiss, who looked at a small asbestos paper factory. But that's largely a wet process and he didn't find anything much in that. The other was a study by Elwood and Cochrane in England, which again was rather a small factory, and they didn't find...they

A. (cont'd.) had one mesothelioma, but that mesothelioma had actually been exposed to crocidolite and didn't belong to the main chrysotile cohort. So there were no cases, but the studies have been very small ones and were not definitive.

So it seemed very important to examine large chrysotile...cohorts of persons exposed to chrysotile in manufacture, and because I thought it would help to have a contrast, I took these two factories belonging to Raybestos, the one company which had manufactured only chrysotile, and so that I would study another factory in exactly the same way, I decided to study their large third factory, which was the Mannheim plant. I thought it would be more helpful to be able to look at both mesothelioma and lung cancer exposure response in these three plants.

As I say, the interest was particularly as far as mesothelioma was concerned. Then having started to study this factory, I then found Dement had started on the same plant, and I found that the other NIOSH group had started also on the to restudy the Mannheim plant and to look at exposure response, but they are in the middle of doing that now.

But the third factory hasn't been studied by anybody, so that is a very similar factory to the one that Newhouse has reported recently in England.

Q. My last question is switching gears. Yesterday in testimony we had a suggestion that the Commission should consider setting a standard for asbestos mining should mining happen in Ontario again, based on midget impinger sampling, plus fiber sampling, but because of the epidemiology...and I note in your and your husband's 1974 paper there is the suggestion on tab two, on the first page, that in the absence of epidemiological findings based on fiber counts, and lack of a satisfactory means of conversion, particle counts should continue to be used for control in industry.

Q. (cont'd.) Now granted, that's some time ago that you wrote that, but I am wondering whether you would still make that recommendation today.

A. Well, it seems to me that the membrane filter is a very considerable advance, but on the other hand it doesn't give us information which we are able to relate to previous studies which had to be based on midget impinger. What would be wrong with doing both methods of...doing monitoring both with an impinger and with personal membrane filter?

I mean perhaps it's expensive, but it does seem to me that it would be valuable to have the midget impinger information to be able to make comparisons, in view of all this discussion about conversion, with previous studies.

MISS JOLLEY: Thank you very much, Dr. McDonald.

DR. DUPRE: Thank you.

Mr. Hardy, might I suggest that perhaps we take our little break now and then you and the Commissioners will...

MR. HARDY: Fine. Fine, let's do that.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Mr. Hardy, will you proceed, please?

MR. HARDY: Yes, sir.

CROSS-EXAMINATION BY MR. HARDY

Q. Dr. McDonald, we have had a lot of discussion about what you are going to be doing with the exposure information at the Charleston plant in South Carolina, which is plant A in your preliminary report which is tab fourteen. I'm curious to know what sort of exposure calculations you were planning to conduct with respect to the other two plants in that study - plants B and C.

Q. (cont'd.) Could you tell us what you are planning to do with the exposure information there?

A. I was planning to do exactly the same as in the Charleston plant, as far as I can, because the purpose of studying the three plants was to try to make a comparison between the three.

The Mannheim, the data are probably rather similar, but it is complicated by the fact that there's mixtures of amphiboles and chrysotile. I just don't know quite how I'm going to be able to deal with that. But the Connecticut plant, at the moment we've got very much smaller numbers of environmental measurements, but we are just going to have to do the best we can with those.

The range in a friction materials plant doesn't appear to be quite so big.

Q. Could you maybe give us some idea of how much raw data in terms of measurements you have at the two plants? Why don't we start first with your plant B in Stratford?

A. Well, I'm still trying to get the environmental measurements - any more that they may have. There are several studies in the...several sets of measurements in the 1930's, made by Metropolitan Life, there are a few sets of measurements made by the company, but very, very little, and then there is at the moment rather a big gap until the more recent measurements. We have since about 1950, but not very much between.

But the measurements are distinctly scanty.

Q. One of the things that I know Newhouse and Berry did with the friction plant that they recently studied in England was to attempt to simulate conditions in that plant in previous years. Have you considered doing that at this plant in Stratford?

A. I haven't considered doing that. But I think

A. (contd.) we should talk to Skidmore, who was responsible for the environmental side of that study.

Q. You mean Skidmore who worked with Newhouse and Berry?

A. Yes.

Q. I gather you think that that sort of simulation might be a useful way to determine what exposures were in the past?

A. I think it might well be. In fact I think that simulation might well give us some further information on the other plants, too, on the textile process, and the Mannheim plant has a combination of friction and textile.

Q. Could you just give us some idea of raw data on exposures exists at the Mannheim plant?

A. Very similar to those at the Charleston plant. On the whole, they are a slightly higher level, but I haven't really gone into any detail on this, and on the possible mixtures of amphibole and chrysotile in the different department.

Q. I gather from what you say then that there are considerably more measurements existing for the Mannheim plant than for the Stratford plant?

A. Yes.

Q. When you told us before that you would hope to have some results within the next year, does that mean results for all three plants, or just results for the Charleston plant?

A. Oh, I hope to have results from all three plants, but they will have to be based on the best I can do in the way of the environmental measurements that are available between now and early next year.

Q. Another subject I wanted to talk to you about stems from your series of articles where you identified mesotheliomas in North America, and perhaps if we look at tab ten, which is I think the most recent of those articles, I wanted to clarify

Q. (cont'd.) one point.

As I understand what you did, was that through contacting pathologists both in the United States and Canada, you attempted to identify all of the mesothelioma deaths that had occurred in recent years?

A. Well, for the United States I only attempted to obtain deaths in 1972. For Canada I have been doing it at intervals since 1966.

Q. Then once those deaths were determined, one of the things that you further tried to determine was whether the individuals had had asbestos exposure during their lives?

A. Yes. The method of the study was to match the case with a control, with some other cancer which had caused metastases in the lung, and to send out interviewers to locate families and give them exactly the same questionnaire - not telling the interviewers which the cases were and the controls - and to get in that way as comparable an occupational history as we could.

But of course, a retrospective occupational history isn't necessarily, certainly, an incomplete one, but one hoped that it was similarly incomplete in the controls as in the cases.

Q. I think table two, page 1652, provides some of the results of that questionnaire survey. Am I reading that table correctly when I look at the column labelled 'unlikely asbestos exposure', which includes then both cases and controls, when I observe that you found a substantial percentage of mesothelioma cases without any likely asbestos exposure in their background?

A. Yes. But actually this table two shows the results expressed as the...as having done jobs which were categorized definite, probable, possible and unlikely, by various experts in the United States and Europe and Canada, but in fact

A. (cont'd.) that wasn't our main analysis. That was just showing what the results were using that method.

In fact, we thought that the better method was to select out occupations as you see in table three, which had been reported associations with mesothelioma, and so instead of taking the probable, possible and so on, we in fact think that this table three tells us more in that..but you will still see that a lot of cases had done none of these jobs that were reported to have been associated with mesothelioma.

Q. I guess if we are looking at table three, then in particular we are just talking about the hundred and one male cases who are listed across from category G - none of the above?

A. That's right.

Q. From whom you were able to identify no previous occupational exposure to asbestos?

A. Yes.

But also you could add on that category F, which were other jobs which had been listed by these experts as being possible, at least, exposure to asbestos, but whose jobs didn't fall into these more-definitely-associated-with-mesothelioma jobs.

So you can say that a hundred and fifty six of the three hundred and forty-four men, male cases, did not work in one of these trades that were associated with mesothelioma, compared with two hundred and sixty-six controls.

Q. Is it fair to say, based on this sort of evidence, that there are, in the general population, mesotheliomas occurring which appear not to be attributable to asbestos exposure?

A. Yes. We have come to that conclusion from a number of different pieces of evidence. One is that these cases of mesothelioma were reported by pathologists before the industrial exploitation of asbestos could have caused mesothelioma.

A. (cont'd.) You do find when you do national surveys of mesothelioma, a very large proportion that other studies have found, apart from ours. There have been a substantial proportion of people who have not apparently had any exposure to asbestos.

Q. Isn't it true also that there have been clusters of mesotheliomas found in various towns in Turkey, which have been attributed to fibers other than asbestos?

A. That's true.

Q. So that I would gather then that one of the things you have found is that there is probably some sort of general background rate of mesotheliomas for which we really don't know the cause?

A. That's true.

MR. HARDY: I don't think I have any further questions.

Mr. Chairman?

DR. DUPRE: Dr. Uffen?

DR. UFFEN: Just one that's somewhat related to this.

In your tab number eleven, we were earlier on looking at table one on page sixty-five, down at the bottom of the table it says, it lists seventy-two cases from other occupations.

THE WITNESS: I'm not quite with you. What page?

DR. UFFEN: It's page sixty-five, table one.

THE WITNESS: It's not tab...

DR. DUPRE: It's 675.

DR. UFFEN: I'm sorry. It's 675.

THE WITNESS: 675? All right.

DR. UFFEN: When you wrote to the pathologists and identified all these things, it says:

DR. UFFEN: (cont'd.) "Occupational and residential histories were obtained for most of them".

How was that done? How were they classified? Like, were they classified as electrical workers, housekeeping...

THE WITNESS: What exactly was done was as full an occupational history as possible was taken, so that the interviewer would ask the relatives or friends what the person did when he left school, and try to find out the actual job and the industry within which this job, in which he did this job, and try to get the years in which he was doing this job, and go right through his life history in that way.

It was similarly for residence. We asked them where they were born and the changes of residence, and we asked the people to classify them, as a very rough and ready thing, as urban or rural.

DR. UFFEN: Would it be safe for me to assume, or perhaps you could put in a more positive way, of all those different occupations would any of them have had, say more than five cases of mesothelioma, or more than one?

THE WITNESS: I don't understand what you mean.

DR. UFFEN: Suppose in the 1972 other occupation cases there were some electricians. Would there have been, say, more than five electricians or five plumbers, or would any occupation have had more than one case?

THE WITNESS: Well, they would be groups of different sizes, and that's eighty-eight cases in all.

You are referring to the first column of cases? Or any particular...

DR. UFFEN: 1960 to 1978 total cases...

THE WITNESS: Oh, total cases? Yes.

DR. UFFEN: See the figure seventy-two?

THE WITNESS: Seventy-two.

DR. UFFEN: All I'm trying to find out is, apparently eight in shipyards is significant. What would be considered insignificant?

THE WITNESS: Well, this...it wasn't that the eight was considered significant in shipyards. It was that these were the occupations which have been reported to be associated with mesothelioma, so the occupation was selected.

DR. UFFEN: I guess what I'm getting at is, is it possible that there are some other occupations associated with mesothelioma that haven't been recognized up to that time, and which ought to have? And if so, how many cases would you decide was significant?

THE WITNESS: Right. Well, could I answer that in the other paper, the Malignant Mesothelioma in North America, because that's a bigger series and it shows you more how we did it.

DR. UFFEN: Okay.

THE WITNESS: I didn't do that in the Quebec study. If I can find it...oh, ten.

DR. UFFEN: Which tab are we on now?

THE WITNESS: Ten.

You see, we selected these occupations which are the same as those you'll see under the Quebec, in the Quebec paper: insulation, asbestos production and manufacture, heating trades, shipyards, construction industry.

Then we have a group of the other jobs that were listed in...actually it was Selikoff's list which he had built up to identify jobs that he thought entailed asbestos exposure.

Then we have a group of other occupations.

Now, you'll see in table four that we have

THE WITNESS: (cont'd.) put out...I'm just trying to think how they were...we have put out all these occupational groups that were industrial jobs, and we have excluded things like domestic work, professional work and so on. So anybody with an industrial job came into this classification having taken out all the people in those categories I have just mentioned above.

This enabled us to look to see whether there were any other occupations which might have...one might suspect of causing mesothelioma, and you would look for an excess of cases over controls. Well, there was a small excess, but they were very small numbers - a factory making rubber products - but, you know, there wasn't anything to hit you in the way of an excess among the cases, which you would expect if there were other easily recognizable jobs that were likely to give rise to mesothelioma.

DR. UFFEN: I may be stubborn on those things that don't count, but factories making rubber products - one of the things they do in a rubber product is they powder them with talcum to keep them from sticking.

THE WITNESS: Yes, very well.

DR. UFFEN: I used to do it years ago, so I...

THE WITNESS: Your talc or tremolite cases would be...it could be.

DR. UFFEN: I'm getting worried. I want to make it to eighty-five.

THE WITNESS: I found it of some interest that we didn't find any evidence there of garage work, which might have entailed brake lining exposures, giving us any excess. But a negative finding doesn't really mean very much. But there wasn't anything positive.

DR. DUPRE: Dr. Uffen, the Chairman's only concern is that you make it until the time our report is due. After that, you are on your own.

DR. UFFEN: I'm not sure I'll last that long. I only hope to make it until I'm eighty-five.

DR. DUPRE: Dr. Mustard?

DR. MUSTARD: A couple of areas I would like to explore and I'll be doing it with some difficulty...

THE WITNESS: I'll be answering with some difficulty.

DR. MUSTARD: ...because of large elements of ignorance and uncertainty about the total dimensions of the data, but why not take tab thirteen and look at table two, and reflect on the data you so neatly put together there.

As one looks at it, one can ask a variety of questions, and I realize it is set up for a very specific task of looking at the ratio of respiratory, digestive cancer to mesothelioma, but it's fascinating to see the size of the cohorts traced, and the number of deaths. You suddenly get a perspective that you never had before...at least we've never had before.

THE WITNESS: Rather pathetic, isn't it.

DR. MUSTARD: Then you come to this question about numbers, and one wonders a little about the interpretation.

The second thing that comes up, of course, is a wide range of exposure histories in these numbers. Some of them are a group of four hundred people with intense exposure, and some of ten thousand with a range of exposures.

But if you go through it, recognizing those limitations, some things startle you - at least startled me - and maybe you could help me sort them out.

For example, if I take the two chrysotile mining studies - take the three chrysotile mining studies - you have about one cancer death in excess of what you expected, for two hundred people in the cohort.

That's an approximation, but that's about what

DR. MUSTARD: (cont'd.) it works out to.

The Nicholson study is one to thirty, and that's perhaps in keeping with the fact it's maybe a much more-exposed cohort. I suppose that would explain it that way.

THE WITNESS: A subcohort of our cohort, and comparing them they are exactly the same for the same exposures.

DR. MUSTARD: Yes. So that explains that one.

Then I come to the one from Rubinno, and there are no lung cancer deaths in that one. That's good and I suddenly sort of say, well, what was going on in that study. That stands right out and...is the exposure different or what happened? Or did they all smoke fifty packages of cigarettes a day and maybe you couldn't show anything else anyway. Do you know?

THE WITNESS: Well, I'm speaking off the top of my head, but I think they did have a log of lung cancer and a lot of smoking in that part of Italy.

Well, they had a lot of lung cancer in that area, and I think that's the way the expectation is calculated.

DR. MUSTARD: I see.

I'm going to skip the next two, particularly talc, because my colleague asked questions about that, and skip...because I'm not interested in talc on babies, I'm not a pediatrician...turn to the crocidolite mine and I would be fascinated to know whether the misprint is in the twenty-two point six or in the five point four.

THE WITNESS: I would too.

DR. MUSTARD: Because if the misprint is on the twenty-two point four, then the lung cancer incidence is one to about eight hundred.

Now, with crocidolite, which is the only...I guess, crocidolite mining operation...and that's a very large cohort of people, and that stands out and I wonder if you probe that what you might find.

THE WITNESS: One of the complications about all these is the length of observation, and have the people lived to the time in which they get lung cancer.

DR. MUSTARD: So the only two that really look attractive are the McDonald study and the Nicholson study, because they reasonably stand out and they really do demonstrate the exposure history phenomenon in terms of selecting the cohort.

But still recognizing all that, I find it fascinating when I sort of look at the lung cancer question.

Then when you go down to the Newhouse and Berry 1979 study, which is a mixed exposure, there they have about one lung cancer per fifty people in the cohort.

Then I was trying to rack my brain as to what the next one represents, because in the eight thousand cohort they have virtually no lung cancer.

THE WITNESS: Well, that's this latest friction material plant that Newhouse and Berry studied...largely chrysotile, but they had crocidolite for a limited period. So it had to go down as a mixed exposure.

DR. MUSTARD: They had eight mesotheliomas in it.

THE WITNESS: They had eight mesotheliomas, all of whom were exposed to crocidolite.

DR. MUSTARD: But the lung cancer, you know, again you may say that maybe the exposure history, how long they were exposed...but my problem as I go through this and look at these things...

THE WITNESS: Yes. Well, in friction materials plants the exposure levels are generally quite low, and a lot of the process is wet. There are certain processes which, in preparation and in...there is a dry friction material process, but they are the minority of the people that are doing this.

DR. MUSTARD: Okay. That helps me a little bit

DR.MUSTARD: (cont'd.) with that. It doesn't help me with the ones that have large cancer rates, for example the Henderson and Enterline study looks like it's...again, I calculated what it was working at, but it's a much higher proportion.

THE WITNESS: Yes. You see, I'm afraid you have to recognize that this is an awful hodge-podge, the Henderson and Enterline study is a retiree study in which they have eliminated people before sixty-five, and in fact that was why they had they had very few mesothliomas, because they were losing them before sixty-five.

DR. MUSTARD: So one could sort of take this epidemiological work - admittedly it's a hodge-podge - but in a sense you could probably start shifting them around and come up with some relatively interesting perspectives.

In other words, there are obviously going to be a number of variables, but I think you are helping me sort it out.

Has anybody done that? Has anybody just taken all this and looked at them from the standpoint of exposures, cohort, how long they have been exposed, and try to sort of look at what the data means in terms of the different studies?

What I'm trying to get at is, we had an epidemiologist speak to us in October or November, by the name of Sackett, who tried to tell us that when you are looking at epidemiological data it's all basically weak, but what you start to look for is consistency in observations to give it strength.

My problem is, how do you take all this and try to just shift it around so that you are looking at oranges and oranges in each of the sectors, and see if the oranges and the apples and the thumb tacks have some degree of consistency.

THE WITNESS: Well, I mean, as you rightly say, this table was put together for one specific purpose, and that was

THE WITNESS: (cont'd.) to look to see whether there was any way of estimating the mesotheliomas, the number of mesotheliomas...well, sorry, the proportion of lung cancers which were attributable to asbestos, by looking at the ratio between lung cancer and mesothelioma, and it would be only by taking out the studies and doing a comparison...now, Nicholson did a paper for the Swedish government - I don't know whether you saw that paper...

DR. MUSTARD: Yes, I have.

THE WITNESS: ...in which he did try to do this epidemiological-type analysis. I don't think that it was always as objective as it might have been, or even as accurate as it might have been. It's very difficult to do this, but that has been the approach.

DR. MUSTARD: Let me just say one other thing from looking at this. The insulation mixed, in terms of your ratios, just from my elementary course in how to appreciate the data in this sort of thing, shows a remarkable consistency, doesn't it, in your ratios that insulation using mixed fiber...

THE WITNESS: Yes.

DR. MUSTARD: ...you really do show a very good relationship, or a consistent relationship I should say...

THE WITNESS: With excess lung cancers.

DR. MUSTARD: ...in mixed insulation.

THE WITNESS: Yes, yes.

DR. MUSTARD: That's a remarkably consistent relationship?

THE WITNESS: Yes, it is.

DR. MUSTARD: Well, that helps me a little bit on that problem. I don't think that this Commissioner, anyhow, will become an expert on how to sort all that out, but I'm very grateful for being confronted with that table and all the questions it raises, as well as the points that you tried to make.

THE WITNESS: Well, I understand that a lot of epidemiologists are baffled by this. I understand that Professor McMahon from Harvard has been looking at the question and finds it all very difficult to draw conclusions. I don't think you are alone.

DR. MUSTARD: Now, could I go back to tab fifteen? And I would like to ask some questions that in a sense have been covered, but I want to see if I can make certain I understand exactly what has been done in the study.

First of all, the samples were sent to you by a pathologist, is that correct?

THE WITNESS: That's correct.

DR. MUSTARD: Who was asked to send another sample as a control?

THE WITNESS: No. The controls were the carefully selected controls that we had in our study. Somebody visited, myself or another colleague who was working on this project, visited each hospital and collected information about the cases of mesothelioma, and at the same time selected from the pathology files a control matched for age, for period in which the death occurred, the sex, and we selected cases of pulmonary metastases from some tumor outside the chest.

So this was a case in which there wouldn't be...a control in which there wouldn't be any reason to think there was an association with asbestos exposure, because there was a mixed group of different primary tumors.

DR. MUSTARD: And there was convincing evidence that the primary was somewhere else? It wasn't case of a lung cancer...

THE WITNESS: The primary was identified...no.

DR. MUSTARD: .... metastasizing to another site?

THE WITNESS: No, they were all identified primaries outside the chest.

DR. MUSTARD: Now, I'm sure you are aware that if you take a cancer, particularly a relatively undifferentiated one, and send it to five pathologists, there is a reasonable probability you will get three answers?

THE WITNESS: Yes.

DR. UFFEN: Not five?

DR. MUSTARD: Pardon?

DR. UFFEN: Not five?

DR. MUSTARD: No, I'm talking about three.

I don't know if that would be a problem or not, but it's a bit worrisome, in other words, how completely one can exclude that the primary does not belong, that it's secondary someplace else.

THE WITNESS: What are you talking about? The...

DR. MUSTARD: I'm just trying to make sure that the control, which was a secondary tumor in the lung, that the probability of that in your mind is perfect, that none of those cases could actually have been a primary in the lung that had a secondary in another site, in which the pathologists had reversed the process - an experience I have had in my lifetime.

THE WITNESS: Well, I wouldn't exclude the possibility there might have been one, or there might have been two. But these were pretty clear cut, you know, real tumors, gut tumors, and I don't...I think it's very unlikely that there could have been more than one that was incorrect.

DR. MUSTARD: Okay.

The second question to this, when the individual examined the tissues to make the fiber estimates, they identified the fibers on the diameter-length ratio question?

THE WITNESS: Yes.

DR. MUSTARD: And then they did an analysis to

DR. MUSTARD: (cont'd.) try and determine fiber type?

THE WITNESS: Yes.

DR. MUSTARD: Did they record any other information about the fibers?

THE WITNESS: Well, I think Pooley has recorded the length and we've never gone into this. We've never looked. I think he has something on the length distribution...

DR. MUSTARD: Length and diameter?

THE WITNESS: It's possible. I'm just not sure. But we haven't analyzed this. We've only just, so far, done this type analysis and weren't taking account of the length and the diameter.

DR. MUSTARD: Because that now takes me to table one, again tab fifteen...

THE WITNESS: In where?

DR. MUSTARD: Tab fifteen. It's the same tab as you were in.

THE WITNESS: Oh, yes. Table...oh, I see, that one. Yes, I've got it. Yes.

DR. MUSTARD: Let me see if I have got a grasp of this table now. For the males there are seventy-six controls and seventy-six individuals with mesothelioma.

There has to be a misprint then under anthophyllite, because I think it adds up sixty-six, and I presume there's either sixty-one or twenty-three for...

THE WITNESS: Yes.

DR. MUSTARD: Is that correct?

THE WITNESS: That's fair. It's awful.

DR. MUSTARD: Well, at this Commission there's time to add up when we are sitting up here. We like to show that some of us can add.

DR. MUSTARD: (cont'd.) Now, one of the things that I guess I get the information from table two, is a tissue could have chrysotile and amosite and crocidolite in it, or could just have chrysotile in it, by looking at the distribution of the table.

If I go to table two, I think there are twenty-nine of those sixty that are listed there - I think that's what the addition comes out to - that had either amosite or crocidolite in them, more than one by ten to the sixth fibers per gram of dried lung?

THE WITNESS: These are not exclusive groups.

DR. MUSTARD: That's what I'm trying to get...I see.

THE WITNESS: The person could have been in more than one of those occupations. It's stated in the text.

DR. MUSTARD: What I'm trying to get at, really, is that - back to table one - that the...as I add up the cases, I'm trying to determine of your seventy-six samples, how many of them had only chrysotile fiber alone in them.

Is that true for any of them? Can I make that calculation somehow or other?

THE WITNESS: Had only more than one million fibers of chrysotile, only? Yes. Well...

DR. MUSTARD: Well, if we wanted to say greater than ten? Greater than ten?

THE WITNESS: Greater than ten?

Well, there are only twenty-six cases, or twenty-seven cases.

DR. MUSTARD: There's twenty-seven cases, and what I'm trying to say is, I go down to the right now, does that mean I have some mesotheliomas with only chrysotile in the lung?

THE WITNESS: There would be some, yes.

THE WITNESS: (cont'd.) I can tell you the overlap situation on the amosite and crocidolite, and in fact it is stated in the text.

DR. MUSTARD: That's right.

THE WITNESS: There was one man who had only crocidolite, and therefore fourteen had amosite and crocidolite.

Therefore, taking away the amosite people who had more than one million fibers, there were twenty-six. So twelve had only amosite.

DR. MUSTARD: Okay. Twelve had only amosite, nothing else?

THE WITNESS: Oh, no. They had some chrysotile, probably, as well.

DR. MUSTARD: Okay.

Well, let me ask the other question, pose another hypothesis that one might put forward from table one, because I guess with most data as a scientist you are trying to think of an alternative hypothesis, and some of them are pretty crazy, and this one I'm sure will sound like that.

But the one fiber that stands out as being in these lung tissues, including the controls, is chrysotile.

THE WITNESS: Correct.

DR. MUSTARD: Now, the hypothesis that this suggests that chrysotile may not be important is based on the fact the control shows essentially the same distribution.

But suppose that the fiber dimension in the controls was significantly different from that in the case, and suppose I wanted to build an argument that in effect the fibers in the controls are indeed chrysotile, but they are of a dimension which is different from that which is associated with the cause of mesothelioma, and the fibers in the cases are indeed of that dimension.

DR. MUSTARD: (cont'd.) I would take it, on the basis of the information you have given me, that's a tenable hypothesis. It may be very unlikely, but it's a tenable hypothesis at the moment because there is no evidence against it, and the probability of my being right is zero, but ...

THE WITNESS: Well, I mean, what we are looking at here is the fibers in the lung tissue, and I mean, even if you are...it is possible that you could get that difference, but then you would have to further and say well, that is likely to be the same in the pleurae as it is in the lung.

DR. MUSTARD: I realize that the next thing is to go and collect the pleurae and redo the whole analysis, that one could?

THE WITNESS: Yes.

Well, I think we ought...I must Pooley to do an analysis of these by fiber size.

DR. MUSTARD: Because one line of evidence that we have heard is that the dimension of the fiber was very important as to where it distributes itself in the pulmonary tree, and that this maybe be important as to whether the fiber then penetrates the tissue and works its way through to places like the pleurae.

The reason I bring that forward...and I'm not sure that you were surprised to see the amosite and crocidolite distributions...but I would have thought that they would have been a bit...

THE WITNESS: Well, I was rather surprised.

DR. MUSTARD: ..stronger than they are.

THE WITNESS: Yes.

DR. MUSARD: Particularly in light of the other nagging thing that worries me about this table, and that is what I had begun to conceive as being a fact, and it may still be a fact, and that is that among these fibers, chrysotile is the one that

DR. MUSTARD: (cont'd.) tends to disappear from lung tissue, that either the Ph affects it or the clearance mechanisms get rid of it.

In a sense, if one is building an association between other asbestos fibers and mesothelioma, and chrysotile was supposed to be the one that's cleared, I find the relationship they show in this table surprising.

Is that unreasonable, to be surprised at that?

Have I made myself clear about my problem in terms of the clearance story and the associations that are here?

THE WITNESS: In terms of the fact that a large proportion of the chrysotile that is inhaled is exhaled, goes out again, you mean?

DR. MUSTARD: No, and also I think we have been under the...at least I have been under the belief, which is not to include any of my colleagues in this assumption...I have been under the belief that chrysotile fibers tend to disappear, with time, from the tissues. They are either cleared from the tissues during the years that the macrophages can handle them, or they undergo dissolution because of Ph effect, or something along this line.

THE WITNESS: Yes.

DR. MUSTARD: And because of that information, I again would have expected here to find the chrysotile content to be less than that for the other fibers - particularly in association with mesothelioma.

THE WITNESS: Well, I think that in fact when you think that more than ninety-five percent of the asbestos that is used is chrysotile, you expect to find a lot more chrysotile in the lungs.

I would also say that you know that the chrysotile goes out to the pleurae. You know that there must be a lot of

THE WITNESS: (cont'd.) chrysotile that goes out to the pleurae. I mean, how otherwise do you get the pleural thickening that you get when a person has been exposed for a long period of time. It's related particularly to the length of time since first exposure.

DR. MUSTARD: So the argument would be that because there is much more chrysotile used, that indeed these are low figures and maybe one percent of what might have been available if you had measured it at the right time, and for the other fibers it may be ten percent or twenty percent?

THE WITNESS: Well, I think they are coming in and disappearing all the time, probably.

DR. MUSTARD: It's very interesting.

THE WITNESS: Yes. I think it's a lot of chrysotile in the lungs.

DR. MUSTARD: Now, let me move to the next point that comes out of that.

The controls contain a fair amount of chrysotile. Indeed, it looks to be remarkable comparable to that of the cases.

Do you have any views as to why the controls have acquired that much chrysotile? Assuming that it comes in and is cleared, it is suggested the controls, who presumably...maybe you have checked this...did not have an exposure history to asbestos. I'm making that assumption, and may be a wrong assumption. I find it surprising that they are so comparable.

THE WITNESS: Well, quite a lot of the controls do have an exposure history to asbestos. I mean, the exposure to asbestos, occupational exposure, is very common and it's shown in the Mesothelioma in North America. It gives you a picture of the amount of exposure there is to asbestos in the controls.

I think that it is difficult to reconcile this equality of content, of chrysotile content, in cases and controls because you would expect that even if I assumed that, as I do,

THE WITNESS: (cont'd.) that amphiboles are the main trouble in causing mesothelioma, you would expect that they would have got more chrysotile than did the controls because they got it at the same time as the amphiboles.

You saw that we had looked, in this last table, to see whether there was a difference between the low amphibole and the high amphibole in their chrysotile content, and we didn't get anything out of that. That all looked equal.

DR. MUSTARD: You see, I would have expected the chrysotile control distribution to be shifted downward compared to the cases, in that table one. That would have been my expectation.

THE WITNESS: Well, the study was...at the same time as Pooley analyzed these, he analyzed a case control study of mesothelioma from the United Kingdom, and he found essentially the same result. Or if anything, it was the other way around - that the cases had less chrysotile than the controls did. They weren't as carefully matched controls as ours were.

DR. MUSTARD: This then raises me to my other far-out hypothesis on looking at this data.

Each one of us responds differently to an injury stimulus. I guess we escape, in explaining it, in terms of genetic variation among human beings.

Could one not formulate the hypothesis that people can indeed inhale chrysotile fibers and have a high lung content and that only those that are genetically susceptible would be the ones that come down with malignancy, and therefore you could indeed have people who had essentially the same chrysotile content in their lungs and only group of whom will have tumors, and the other group will not, because of genetic variations.

I realize there may be be absolutely no evidence to substantiate that fact, but there are other examples of it in biology.

THE WITNESS: Oh, I certainly agree, and I'm quite sure that susceptibility is very different.

But how do you explain the fact that you do get a difference between cases and controls in the amphibole content, whereas you don't in the chrysotile content?

DR. MUSTARD: I was looking at those differences and I agree that they are there, but then I am troubled by my other problem - that the...and obviously this is a feel for what the content of fibers in the lung tissue really means...and it may be that less than one amphibole fiber per gram of tissue is biologically a very significant quantity in terms of its effect.

If that's the case, then I think one could quite easily interpret it the other way around - and that is indeed it is the amphiboles that are important. That, for example, as you said, that you need a thousand fibers of chrysotile to kick the cancer system into action, per gram of tissue, and you only need one of the amphiboles. Then I think the distribution, if you had it, would be fitting it in, in other words, in terms of the impact on the tissues, and I think that is certainly an equally plausible hypothesis.

THE WITNESS: I think there are plenty of hypotheses that are plausible, based on all this data. But to me, if you put together the tissue, this information about amphiboles with such cohort studies as we have with persons exposed to different fiber types, it does have a certain...it does have some consistency.

DR. MUSTARD: But I guess really the thing that we now are enormously dependent upon is the item of research you have been discussing most of the afternoon and were discussing early this morning - further analysis and acquisition of data of manufacturing sites in which chrysotile alone has been used,

DR. MUSTARD: (cont'd.) because essentially until we can build up the same amount of data as we have for the other area, the weighting is a bit out of proportion in trying to make a determination whether the basic data that you have from the mining, and the limited manufacturing data, is compatible with chrysotile being a lesser stimulus for mesothelioma development.

THE WITNESS: Yes.

DR. MUSTARD: But there is that uncertainty in the manufacturing data, that the bulk of the data is from the mixed and we really don't have the same weighting of chrysotile evidence.

THE WITNESS: Right.

DR. MUSTARD: Is that a fair interpretation?

THE WITNESS: Yes, it's a fair interpretation. It's the reason I undertook this study, so I could get some more information.

DR. MUSTARD: Thank you very much. You have answered all my questions and helped me wander through this to sort out the possible hypotheses.

THE WITNESS: Yes. I think there are innumerable hypotheses, and I think we can just choose our own, that we think are the most generally...sort of generally fit the data best. That's all we can try to do in epidemiology.

DR. DUPRE: Dr. Uffen?

DR. UFFEN: Could I ask you a question that just sort of follows from that?

In tab fifteen where we were talking about North America, both the...maybe I'm just getting lost now...were the controls people who had died not of mesothelioma...could you remind me how the controls were chosen again?

THE WITNESS: They were chosen as people from the same pathology in files, who had secondary lung or pleural cancer -

THE WITNESS: (contd.) lung cancer it was mainly - from a known clear-cut primary tumor outside the chest, and who were the same sex and age and died around the same time.

We took the closest person who fitted certain criteria.

DR. UFFEN: I guess my question as a layman is, I wanted to know what is normal and what is abnormal? Could you not have the pathologist tell you what is the chrysotile content...or take their samples and find out...what is the chrysotile content of people who have died not from cancer, not from lung ailments, but from cracked skulls, or something like that?

THE WITNESS: Pooley has got that data.

Has Pooley been here?

DR. UFFEN: No. But we have been given references, so presumably we can hunt it up.

THE WITNESS: Yes, but you actually have his paper that he gave in the same meeting as I gave a preliminary paper here. I think it was...no, it was in this meeting, it was in the...no, it was in the Lyon meeting.

DR. UFFEN: Is the chrysotile content of lungs of other people who died of other things of the same order as in your table one here, that we have been looking at?

THE WITNESS: I think that the...oh, dear, I'm speaking from memory now...I think that in England the chrysotile levels were a bit lower than they were in the American population.

But the...he certainly had controls who died of strokes and things like that, and I don't think they differed from the rest as far as I can remember.

But maybe it would be better to look at the paper and discuss it with Pooley.

DR. UFFEN: Thank you.

DR. DUPRE: Dr. McDonald, just a couple of questions. You...several of your tables, I think, focus on the insulation workers, among others...and a number of your papers, for example tab eight, comment on the high relative hazard of insulation work, and taking an example at random, your comment on...in tab eight on page 448, just before the table.

Now, I just wanted to ask you about one school of thought that has come forth in the course of the summer...the short, sharp burst of exposure school of thought.

Do you give any credence to the short, sharp burst hypothesis as perhaps telling us a little bit why insulators as an occupational group would show such high numbers?

THE WITNESS: I think that would only be conjecture, because I think we have very little information about exposure levels in applying insulation, and certainly when spray insulation was used, I feel sure that exposures must have been extremely high.

Certainly epidemiologically we have absolutely no means for distinguishing between the short, sharp or the continuous, low, and I think you would just be the realms of speculation.

You see, I think those insulators have probably all had pretty heavy exposures.

DR. DUPRE: One other very minor question for my own information. In that same tab eight, as a matter of fact, you opened the article with a very nice summary, meticulously footnoted, of some recent findings about mesotheliomas.

It's just your last statement in the first paragraph which is unfootnoted:

> "There is not a reasonable concern that manmade mineral fibers may not be free of risk under certain circumstances."

Do you have a particular handy reference, as you

DR. DUPRE: (cont'd.) do in the other statements, that...?

THE WITNESS: No, there is not a reference at all. It's just from...it's just because this has been observed in Turkey, and because fibers of very fine dimension are being used, that we I think it is a concern that they may not be free of risk, but not any evidence that they do have any risk. In fact, there is evidence accumulating and it certainly looks as though the exposures to date haven't given rise to any trouble...although it's still rather a short period of observation.

DR. DUPRE: Just one last question, Dr. McDonald, that is going to take you once again back to tab fifteen, table one, and it's going to be the same question as Dr. Mustard asked, but asked by a very crude layman, who is going to put the question this way: One hypothesis that very much ran around this summer, from a couple of expert witnesses, was to the effect that chrysotile asbestos, because its chemistry makes it less acid-resistant than other fibers, is more likely to dissolve in the lung or in human tissue.

Now, to ask my layman's question crudely, doesn't your chrysotile fiber counts that appear in table one, don't these counts shake that hypothesis to its foundation?

THE WITNESS: Well, I suppose that you would...if a lot is disappearing and you are still left with this amount, it does seem rather horrifying, if that's what you mean.

I mean, I think that we just don't know what is happening to the chrysotile. I think it is likely that it is disappearing, and therefore I don't feel that this evidence is in any way, can be in any way definitively to say this bit of evidence alone couldn't tell you that the risk of chrysotile exposure is negligible.

THE WITNESS: (cont'd.) But...because of that fact, it's just that if it is disappearing fast, then there may well be some difference in the time at which these cases and the controls were exposed.

You couldn't take it too far. But you could just say that this would be consistent with the fact that chrysotile wasn't causing a risk, but that's all.

DR. MUSTARD: Let me see if I understand what I thought your answer to my question, which is similar to his, was.

My understanding of your answer in response to that question is that chrysotile indeed might be disappearing at a rate four times as fast as the other asbestos fibers, but because there is far more chrysotile used, then you could have a situation where every amphibole fiber that you inhale, you inhale two hundred of chrysotile, and therefore the rate of disappearance of chrysotile was, you know, four times of that amphiboles, you would still end up with a net higher appearance.

Did I misunderstand your answer?

THE WITNESS: No, I think that's true. I would agree with that.

DR. UFFEN: It doesn't tell you what isn't there.

DR. DUPRE: Well, indeed I'll try this out on you as a layman's hypothesis. I'm looking again at table one, and I look at the cases that all had between ten and a hundred fibers of chrysotile, amosite, crocidolite, and if I said to myself, well, maybe there's some of these mixed-exposure cases in there in which indeed the mixture to which they were exposed was ninety-seven percent chrysotile and three percent crocidolite.

I suppose that my fiber-dissolution hypothesis comes out from this table relatively unscathed, because the number of crocidolite fibers is more than three percent the number of chrysotile fibers, and whatever the difference is possibly would

DR. DUPRE: (cont'd.) have been dissolved?

THE WITNESS: It's compatible.

DR. DUPRE: It's only speculative.

THE WITNESS: Yes.

DR. DUPRE: But it's not lunacy to put it forward? So long as I stay outside of...you've got to stay away from lunacy.

Counsel, do you have any final questions?

MR. LASKIN: No final questions, Mr. Chairman.

DR. DUPRE: Well, Dr. McDonald, thank you so very, very much for being with us today. Welcome back to Canada.

Given what I hear about some of your timetables for your forthcoming studies, it's not unlikely that we may want to make sure that we are writing the final draft of our report with an open line to Montreal, in case you have some results that may enlighten us with it. Thank you so very much.

THE WITNESS: Thank you for having me, and I don't suppose it has been of very much use, but I have been very happy to be here and I suspect I'm being rather hopeful about when I'm going to get results. You have to be hopeful, otherwise you may not get there.

DR. DUPRE: We are also hopeful about a number of things in writing the final report. Thank you again.

Now, I take it, counsel, that we now rise sine die or sine die, depending on which Latin pronunciation one prefers, correct?

MR. LASKIN: Let's just say that the summer portion of the summer school has come to an end and we will resume in the fall term.

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THE INQUIRY ADJOURNED

THE FOREGOING HAS BEEN PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

Edwina Macht

EDWINA MACHT